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Labyrinthine window rupture

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Introduction

SOME patients presenting with sudden sensorineural deafness have suffered a rupture of either their round or oval windows. It is the object of this paper to discuss the diagnosis of these and to present cases which illustrate the place of surgery in their management.

Morrison and Booth (1970) have pointed out the importance of distinguishing between a nerve and a cochlear lesion in cases of sudden deafness where there is enough residual hearing for audiometric evaluation. The nerve lesions appeared to improve on a steroid regime. When this group of patients has been excluded there are a few others where the aetiology is clear, such as mumps labyrinthitis, but in the majority of cases the underlying pathology is speculative and as a result therapy has been as varied as it has been unsuccessful.

Blair Simmons (1968) was the first to propose that membrane breaks might account for sudden hearing loss: 'When a patient tells you that he lost his hearing last Wednesday at exactly 2.35 p.m. the chances are that he did indeed have a truly sudden hearing loss.' In young patients with no previous ear trouble he postulated that membrane rupture was the likely cause, particularly if physical activity at the time was such as to raise cerebro-spinal fluid pressure. Shortly afterwards Fee (1968) described cases where, following head injury but without fracture, there were oval-window fistulae demonstrated at tympanotomy. Goodhill (1971) confirmed by tympanotomy that the oval window could rupture with sudden exertion. He considered that the sudden raising of cerebro-spinal fluid pressure was transmitted to the perilymph by the cochlear aqueduct and described this as the 'explosive route'. He theorized that such ruptures might also occur

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by an 'implosive route' from sudden increased air pressure in the middle ear. Freeman and Edmunds (1972) later reported cases of divers in whom sudden deafness could be accounted for by such implosive ruptures. They later explored such a case with bilateral deafness and repaired both round windows with hearing restoration.

No reports concerning labyrinthine window rupture have appeared in British literature. However, now that the possible benefits of surgery have been shown, some therapeutic guide-lines are needed. It is hoped that the following cases will contribute and indicate the importance of considering the possibility of labyrinthine window rupture when confronted with a case of sudden sensorineural deafness.

Case reports

Case 1: Patient P.B., aged thirty-eight, a company director, returned from abroad by aeroplane. The following morning he awoke with deafness and tinnitus in the left ear. Two days later he developed rotatory vertigo. This soon subsided. An audiogram at that time showed a left-sided sensorineural deafness (Fig. 1). There was no spontaneous nystagmus.

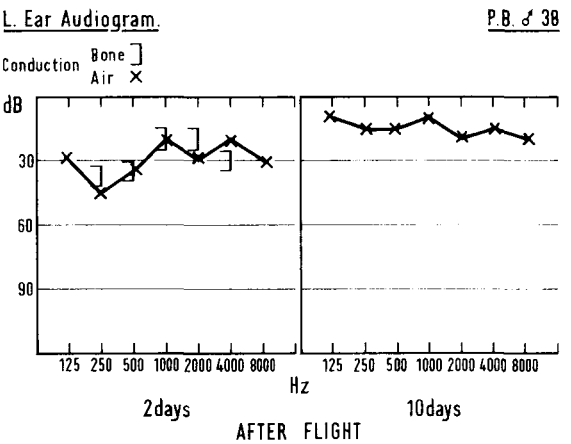


FIG. 1.

He was instructed to rest at home and the vertigo and deafness improved during the next week (Fig. 1). He now occasionally experiences tinnitus but there has been no further vertigo or deterioration in his hearing. No operative procedure was necessary.

Comment

It is conjecture to say that his deafness and vertigo were due to a labyrinthine rupture as no operative proof is available. But in view of the history of possible barotrauma the diagnosis is likely. The hearing recovered with no treatment, apart from rest at home.

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Case 2: Patient A.F., a thirty-one-year-old female, suddenly became deaf in the right ear while descending in an aeroplane. The deafness was associated, at that time, with otalgia and a few hours later, with otorrhoea. Two days later, when first examined, she had the signs of acute otitis media with a perforation and discharge. An audiogram revealed a severe sensorineural deafness with a super-added conductive component (Fig. 2). The latter was thought to be due to the otitis media.

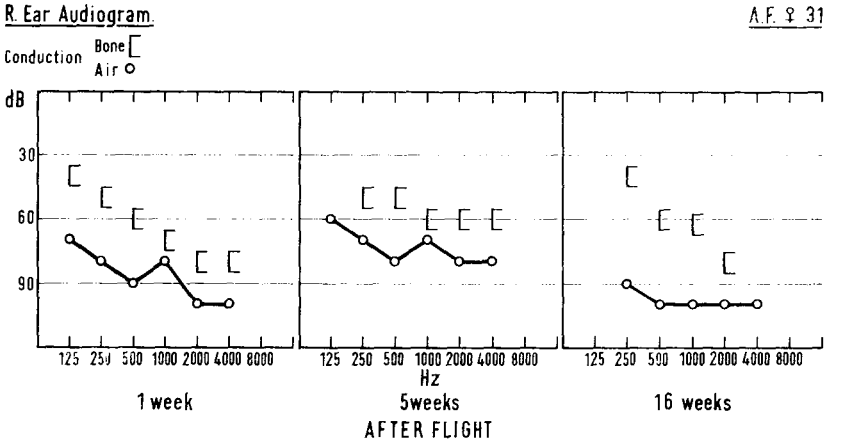


FIG. 2.

The otitis media healed rapidly and the perforation closed spontaneously with systemic antibiotic treatment. The audiogram showed improved hearing (Fig. 2).

The hearing remained static until eleven weeks later when she developed increased deafness and tinnitus and experienced one bout of severe vertigo which lasted for six hours. There was no infection. An audiogram confirmed the deafness (Fig. 2).

With the history of barotrauma, progressive sensorineural deafness and vertigo, labyrinthine window rupture was suspected. An exploratory tympanotomy confirmed this diagnosis.

Operation

A Rosen's anterior tympanotomy was performed. The round window was exposed and its membrane was absent (Fig. 3). Perilymph was seen to be leaking from the round-window orifice. The defect was repaired with a fat autograft taken from the patient's own ear lobe. The mucous membrane around the window was elevated and inverted into the opening and the fat placed over this to act as a plug. A second layer of fat was positioned on top of the first to make contact with the raw surfaces of the promontory. The tympanum was closed as in the operation of stapedectomy.

Post-operative course

On the first post-operative day, the Weber response was heard in the operated ear. A bone-conduction audiogram showed improved hearing (Fig. 4).

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During the first week the vertigo subsided and the hearing showed further improvement (Fig. 4).

This rapid improvement was continued over the succeeding weeks until six months later the hearing is now nearly normal (Fig. 4).

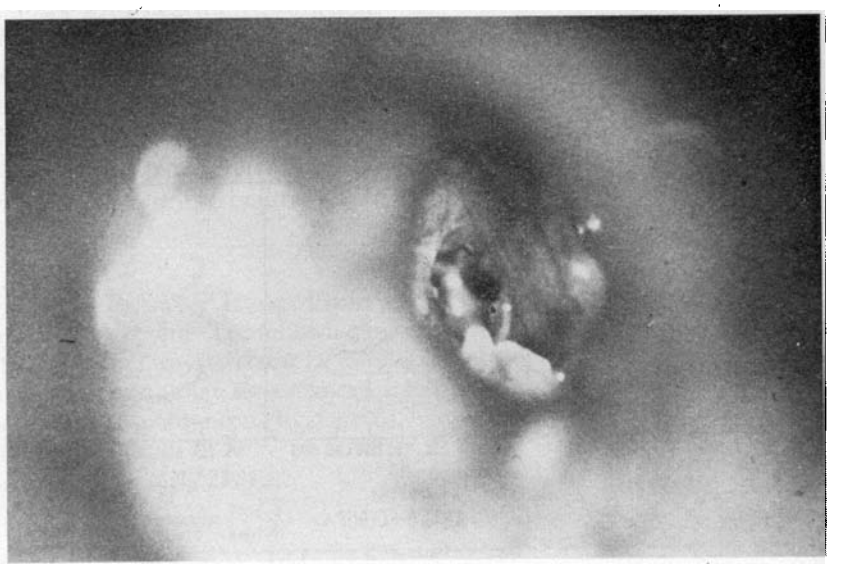


FIG. 3.
Photograph demonstrating round-window defect at operation in Case 2.

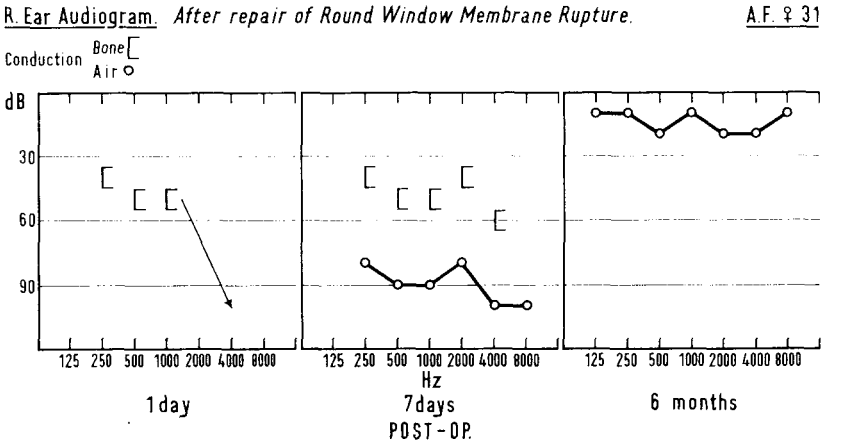


FIG. 4.

Comment

This case demonstrates sudden onset sensorineural deafness due to round-window rupture associated with barotrauma. It was not until vertigo developed

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that the diagnosis was considered and subsequently proven at tympanotomy. The closure of the round-window defect in the right ear provided restoration of hearing and relief from vertigo. It also eliminated the risk of meningitis.

This patient was lucky to have recovered her hearing after such a long interval and it was remarkable that her hearing improved so rapidly after operation. In the light of further experience we would now perform a tympanotomy much sooner. During the delay there was a high risk of irreversible cochlear damage as is demonstrated in the following case.

Case 3: Patient L., a twenty-six-year-old Chinese male, was diving at shallow depths (15 ft.) with snorkel and mask. On surfacing he experienced severe vertigo and a sudden deafness in the left ear. He felt that the ear was 'full of water' which he was unable to remove. There was no recovery within two weeks.

Four months after the initial episode, he still had paroxysmal vertigo. An audiogram revealed a profound sensorineural deafness in the left ear. With the history of barotrauma, sensorineural deafness and paroxysmal vertigo, the diagnosis of labyrinthine rupture was suspected.

Operation

A left exploratory anterior tympanotomy was performed. Apart from two strands, the round-window membrane was absent. No perilymph was seen to leak out after several minutes. The defect in the round window was repaired with a fat autograft from the patient's own ear lobe, as in Case 2.

Post-operative course

The vertigo ceased immediately and has not returned. However, the hearing defect has only improved subjectively. No hearing recovery is demonstrable audiometrically.

Comment

This case shows a clear history of barotrauma causing round-window rupture resulting in severe sensorineural deafness and vertigo. The vertigo was cured by repair of the round-window defect, but the deafness has persisted.

This failure of hearing salvage may be due to the long time which elapsed between the initial causative episode and the eventual diagnosis and operative treatment.

Discussion

History

In cases of acute sensorineural deafness the diagnosis of labyrinthine window rupture must be considered when there is a definite history of either:

(*a*) *Barotrauma;*

(*b*) *Head injury;*

(*c*) *Unusual exertion*—It is difficult to define how unusual this exertion must be. Stroud and Calcaterra (1970) reported four cases of perilymph fistula. Three of these cases developed

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symptoms following comparatively gentle activity—laughing, washing the car and singing in the school choir. It would appear that only a small rise in intracranial pressure may be sufficient to cause labyrinthine rupture. They postulate that a congenital weakness of the round-window membrane or stapes footplate may be a predisposing factor.

Investigations

Accurate pure-tone audiometry is the crucial investigation. If there is sufficient residual hearing, tests of recruitment should be performed to confirm an end-organ lesion. At the same time it is worth remembering that Blair Simmons (1973) showed that excessive high amplitude audiometric testing may result in further hearing loss. Similarly, in the presence of vertigo an E.N.G. might confirm a peripheral lesion.

Goodhill (1971) found impedance studies unhelpful in these cases. Furthermore, if the ruptured window is healing, changing the pressure in the external, or middle ear, may be harmful. Caloric tests are contra-indicated for the same reason.

Management

There are, as yet, no adequate criteria for deciding the correct timing of surgical intervention. However, some plan is essential when presented with these cases and for this reason the following is suggested.

Conservative treatment

Cases of suspected labyrinthine-window rupture should be admitted to hospital. If they are seen early after the causative event they should be treated like any other cerebro-spinal fluid leak. They should be kept at rest sitting up in bed and their progress monitored by daily pure-tone audiometry. Some will recover their hearing as in Case 1 and no further treatment is indicated.

This period of conservative treatment should be used to investigate other possible causes. Haematological findings may suggest a viral aetiology. Generalized cardiovascular disease should be considered. However, if the history is strongly suggestive of membrane rupture, surgical intervention should not be unduly delayed, remembering that a tympanotomy is the next logical step in diagnosis and is unlikely to cause further cochlear damage even if the findings are negative.

Surgical treatment

INDICATIONS FOR TYMPANOTOMY

1. There must be a history of exertion, barotrauma, or head injury.

2. The hearing is deteriorating despite bed rest. No specific time limit can be set. If the history is definite enough and the hearing loss becomes

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steadily worse over the first week, tympanotomy should be performed. Early repair of the membrane may prevent irreversible cochlear damage.

On the other hand the hearing loss may be so gradual as to show neither improvement nor deterioration. Then an interval of two to three weeks' observation would seem reasonable before tympanotomy. However, Blair Simmons (1973) points out that a week should be regarded as the minimum interval, for fear that further cochlear damage may be sustained if the diagnosis is wrong and also to give the window rupture time to heal spontaneously.

3. Paroxysmal vertigo would certainly constitute an indication for tympanotomy as in Case 3. However, any persistent vertigo beyond the first two to three weeks would also merit exploration as illustrated by Case 2.

4. There may be direct evidence of a perilymph fistula in the form of a cerebro-spinal fluid rhinorrhoea or otorrhoea. If present for more than ten days the leak should certainly be closed.

Summary

Some cases of sensorineural deafness are due to labyrinthine window rupture. Three cases have been presented to illustrate different aspects of diagnosis and management of this condition. The indications for surgical intervention have been discussed. The importance of making the diagnosis is that operation can relieve vertigo and restore the hearing.

Acknowledgements

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