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Clinical records

Round window rupture

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Summary

EIGHT patients suffered sudden sensory neural hearing losses associated with exertion or trauma; all were found at tympanotomy to have round window membrane ruptures with leakage of perilymph.

Operative findings, with audiometric, radiological and electrocohleographic results are shown.

The aetiology is discussed, with particular reference to diving techniques and to the cochlear aqueduct.

Introduction

All but one of the cases described were the only patients in the category 'sudden sensory neural hearing loss associated with trauma or exertion' seen in one ENT Department over a period of three years.

As all eight had obvious round window ruptures at tympanotomy, it is now our policy to recommend exploration within two to three days in all such cases unless the hearing, as tested by audiometry, improves rapidly with bed rest.

Case 1—T.A. age 10 years. August 1978.

A schoolboy stood on the head of a rake, causing the handle to strike his left ear. He immediately became deaf and suffered tinnitus on that side but was not vertiginous. Two months passed before consultation for persistent deafness. There were no abnormal signs. Audiometry is shown in Fig. 1 (1).

At tympanotomy an abnormally shallow round window niche and a prominent round window membrane were found with a large rent in the upper third of the membrane and perilymph in the middle-ear. Perilymph also appeared to leak from around the stapes footplate. The round window was repaired with a vein patch and the oval window was sealed with ear lobe fat.

Case 2—P.W. age 30 years. Accountant. May 1978.

In May 1978 the patient was jogging when, after one mile of accustomed exertion, he felt a pop in his right ear, which then seemed to 'fill up'. He finished his two-mile run but one hour later was vertiginous and nauseated. When telephoning his wife he found he was deaf in the right ear. He was treated conservatively in a local hospital, with bed rest and sedation for one week, and then discharged. His hearing improved considerably during this period.

At consultation six weeks' later, he was still unable to stand on one foot and had a first degree nystagmus to the left. His audiogram at that time (Fig. 1 (2)) showed a high tone sensorineural loss. Tympanotomy on 6 June revealed an abnormally shallow round window niche with an easily recognizable membrane.

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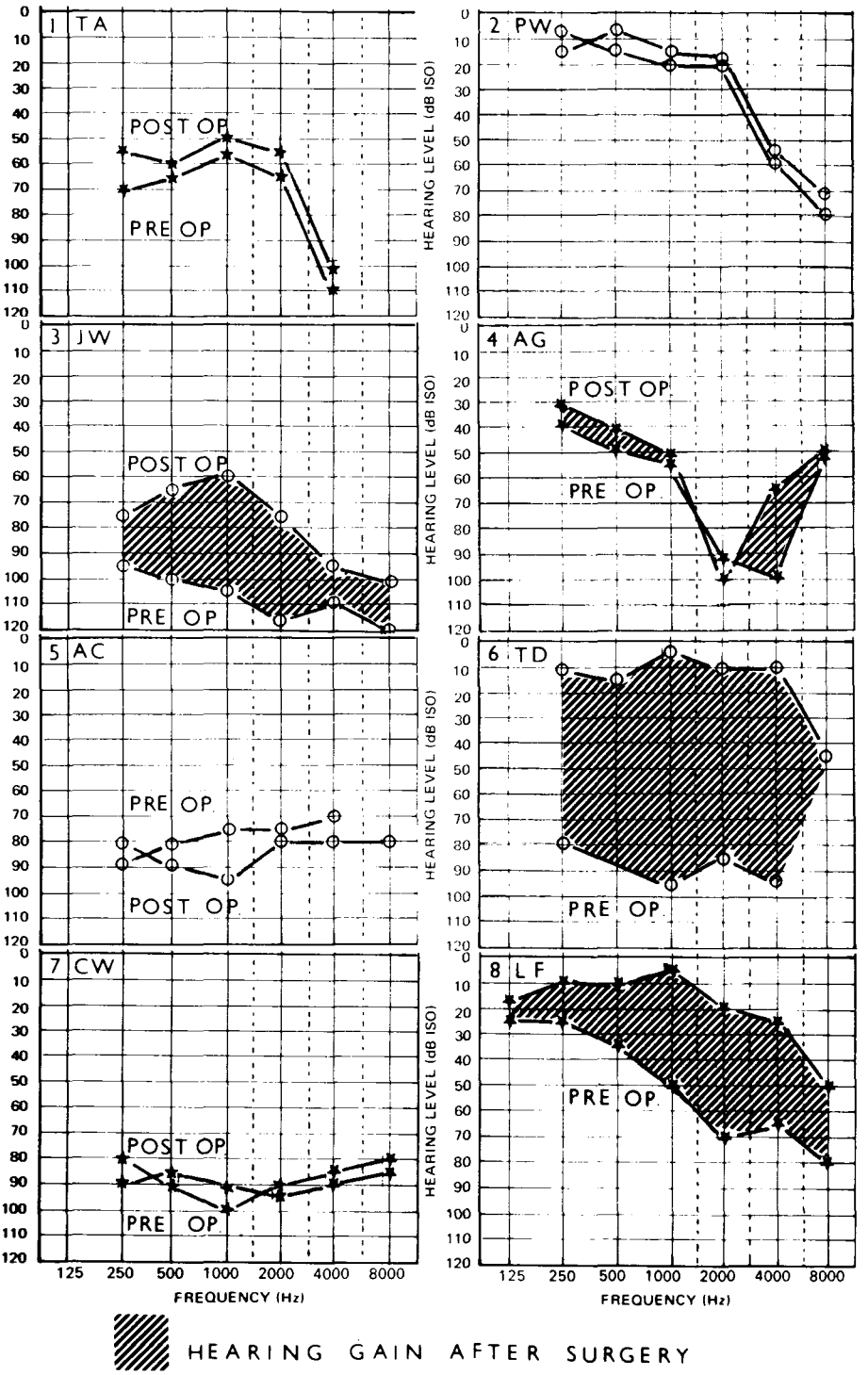


FIG. 1

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This contained a small linear tear, which leaked perilymph at every second stroke of the Manley anaesthetic ventilator, as the central venous and CSF pressures rose and fell.

The defect was repaired with ear lobe fat followed by strict bed rest with sedation for one week.

His hearing seven weeks later was unchanged.

Case 3—J.W. age 44 years. Engineer. August 1978.

Straining to lift a motor mower, this man noticed five minutes' giddiness. He played squash some hours later but the next day awoke with deafness in the right ear and vertigo. As an active SCUBA diver, he knew he had more difficulty in equalizing pressure in his right ear than in the left, and as an engineer he knew that he had a small hearing loss above 5,000 Hertz.

At referral three days later, he had no physical signs other than those of a right-sided severe sensori-neural hearing loss.

After a further three days of conservative treatment with intravenous nicotinic acid, his audiogram (Fig. 1 (3)) was unchanged. Caloric testing showed slight hypoactivity of the right labyrinth at 30° and 44 °C.

Tympanotomy revealed clear fluid in the middle-ear, a shallow round window niche and a prominent membrane with two tears from which blood-stained perilymph oozed when the thorax was compressed. Fig. 2 is a sketch of the surgeon's view at tympanotomy. Both were repaired with ear lobe fat. A period of one week's absolute bed rest with sedation followed.

Case 4—A.G. age 30 years. October 1978.

The patient was lying under a motor car when the supporting jack gave way. His brother had to raise the vehicle to release his head, which was pinned under the chassis. He was unconscious for ten minutes and was then admitted to hospital with multiple facial lacerations and skull fractures. On the third day, he complained of increasing left facial weakness and deafness.

On examination he had a first degree nystagmus to the right, poor balance, a partial left facial palsy and a sensori-neural hearing loss on that side (Fig. 1 (4)).

Tympanotomy showed a fracture in the deep meatus passing through the descending facial canal, stopping at the lateral semi-circular canal. Perilymph was present in the middle ear and ran out of the round window niche and from the margins of the stapes footplate. Two small linear tears were present in the round window membrane.

The leaks were repaired with ear lobe fat and fascia and the patient was put to bed for one week under sedation.

Seven months after the accident no improvement of facial movements on the left side had occurred.

Case 5—A.C. age 41. Amateur diver. September 1978.

Ascending from 110 feet this SCUBA diver felt a pop in his right ear. He felt a little unsteady when climbing back into the boat. The next day he noticed deafness in the right ear with high pitched tinnitus. Any subsequent disturbance of balance was unremarkable.

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Seven weeks' later, he presented to the ENT Department with the same symptoms.

On examination, there was no ENT abnormality except a severe hearing loss in the right ear, with the Weber referred to the left. The tympanic membrane looked normal.

The audiometric investigations are shown in Fig. 1 (5). No improvement was seen when the patient was postured to allow the scala tympani to fill with perilymph (Fraser's test. John Graham, personal communication, 1979).

At tympanotomy, there was a deep round window niche with no visible or palpable remnant of membrane. No fluid was seen until a small leak at the margins of the oval window was sealed with ear lobe fat; fluid then ran out of the empty round window niche. This was plugged with fat supported with Gelfoam, and the tympanic membrane was replaced.

One week's rest with heavy sedation followed.

N.B. Adequate decompression stops on ascent after this deep dive were made. This makes nitrogen emboli unlikely to be the cause of the sudden hearing loss.

Case 6—T.D. age 41. Businessman. August 1975.

A keen amateur diver, this man experienced pain in his right ear while ascending from a depth of twenty feet. He had been breathing compressed air from an aqualung. At the surface, he was able to 'clear' his ears by nasal closure and Valsalva.

Two days later while diving, he noticed deafness in the right ear (Fig. 1 (6)).

Three days later still, he was unsteady, with high pitched tinnitus.

Four years previously, he had had a similar experience in the left ear, diagnosed as a 'ruptured nerve'. A severe hearing loss had recovered spontaneously in this ear after one week, leaving some tinnitus.

On examination, there was no ENT abnormality other than a false negative left Rinne response, with a Weber lateralized to the right; there was no objective evidence of imbalance.

At right tympanotomy seven days after the accident, clear fluid was present in the middle ear. Leaks of perilymph were seen from the round and oval windows, both of which were repaired with ear lobe fat supported by Gelfoam.

Ten days' strict bed rest with sedation followed.

Case 7—C.W. age 51. Schoolteacher. June 1976.

A teacher with a special interest in music suffered a blow to the left side of her head when a heavy blackboard fell from above her. She 'saw stars' and developed a large bruise but did not lose consciousness.

Next day she was unsteady, and deaf in the left ear. On the fourth day, she had tinnitus.

On examination three weeks after the injury, both tympanic membranes were normal. A bithermal Hallpike caloric test showed no response from the left ear. Her audiogram is shown in Fig. 1 (7).

Tympanotomy one month after the injury revealed perilymph in the oval window niche. A rupture was seen in the round window membrane leaking perilymph and the stapes was unusually mobile, suggesting a tear in the annular ligament. Both tears were sealed with plugs of ear lobe fat supported by Gelfoam.

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One week post-operatively, the vertigo had improved with bed rest but the hearing and tinnitus were unchanged at one year, causing considerable misery and incapacity.

Case 8—L.F. age 28. January 1979.

Some hours after a flight on a commercial airliner, Miss L.F. noticed a sudden hearing loss in her left ear (Fig. 1 (8)). The loss did not fluctuate during the three weeks which elapsed before ENT consultation. At this time she had slight tinnitus in the left ear and looking up provoked slight vertigo not present at rest.

On examination, there was no ENT abnormality except lateralization of the Weber to the right at 512 cycles/sec. Hallpike bithermal caloric responses were normal.

Tympanotomy, delayed one week by an upper respiratory tract infection, took place four weeks after injury.

The findings were an easily visualized outward-facing shallow round window niche. The ponticulus and subiculum were poorly developed. A large hole was present in the membrane's inferior portion amounting to one quarter of the area. Perilymph was not seen and the scala tympani appeared dry. No suction was applied to the niche. The hole was sealed with ear lobe fat.

Post-operative audiometry showed a dramatic rise in bone conduction within two days of surgery, the air conduction threshold rising to join it in ten days.

Discussion

If three or four days of absolute bed rest with sedation fails to improve the results of daily audiometry in suspected cases of round window rupture, tympanotomy is indicated.

Many authors with a personal experience of traumatic perilymph leaks have reached this conclusion (Fee, 1968; Stroud and Calcaterra, 1970; Pullen, 1972; Goodhill, 1971).

Fee suggested a diagnostic test with radioisotopes. Stroud tried this, putting technecium labelled albumen in the spinal CSF of one patient with a perilymph leak. No tracer or carrier substance could be demonstrated before or during subsequent tympanotomy at which a footplate leak was seen.

Surgical observations

McNicoll (1978) reported a case of both round window and tympanic membrane perforation. This double injury should be no deterrent to tympanotomy if the hearing loss is mixed, unless both windows can be minutely inspected through the tympanic membrane defect.

During tympanotomy, an abnormally shallow, laterally facing niche is a common finding (Pullen, 1972). Five cases in our series showed this abnormality, and a sixth example in a patient with otosclerosis and round window rupture is reported separately (Barton, Gray and Smith, In Press).

Fee (1968) described three cases and Stroud and Calcattera (1970) two cases, of perilymph leak from the oval window, following trauma or exertion. No comments were made upon the state of the round windows in these cases. It is of interest to note that three of our cases had leaks from both windows, and in one

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case, fluid from the oval window niche had tracked to the round window in the hollow below the subiculum and ponticulus.

The diver who ascended from 110 feet had a round window niche devoid of any membrane and this was initially mistaken for a deep normal membrane until gentle palpation demonstrated a wide open, almost dry scala tympani.

Suction in the round window niche is a possible cause of disruption of the scala media membranes or organ of Corti, especially if the stapes is seen to quiver. Pledgets of cotton wool or foam sponge free of ototoxic preservatives may be used in rapid succession to clear the niche of perilymph or blood, by gentle capillary attraction.

Aetiology

The physical forces on the round and oval window membranes and the expression of these by movements of CSF, perilymph and middle-ear gases are beautifully described by Goodhill (1971). This paper is highly recommended and the exhaustive pathways of pressure detailed therein are summarized here (Tables I and II).

TABLE I

PATHWAYS OF PRESSURE WAVES (AFTER GOODHILL, 1971)

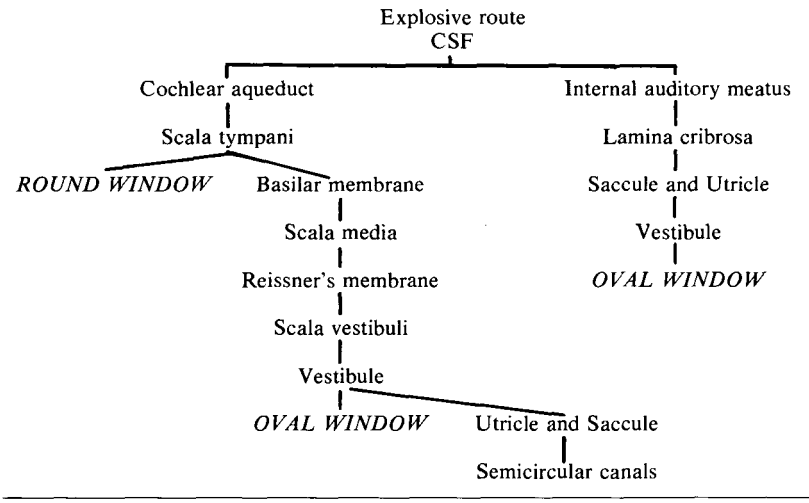
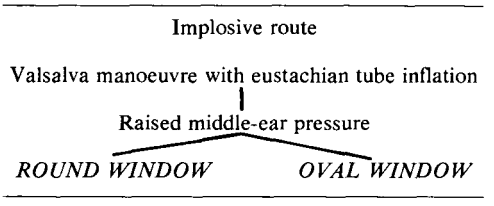


TABLE II

PATHWAYS OF PRESSURE WAVES
(AFTER GOODHILL, 1971)



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TABLE III

HEARING GAIN RELATED TO AETIOLOGY

Exertion	J.W.	Lifting heavy weight	Moderate
	P.W.	Jogging	Nil
Head injury	A.G.	Crushed under engine	Significant
Blow on ear	T.A.	Rake handle	Nil
	C.W.	Falling blackboard	Nil
Barotrauma	A.C.	Ascending from 110 feet	Significant
	T.D.	Ascending from 20 feet	Excellent
	L.F.	Airflight	Excellent

Our patients fall into four groups: exertion, head injury, a blow on the ear and barotrauma. Table III shows hearing gain related to aetiology.

1. *Exertion*

Goodhill's explosive route (Table I) would seem the most appropriate explanation here. Physical effort with chest fixation results in a rise of neck venous pressure and consequent CSF pressure. This may be transmitted by the cochlear or accessory cochlear aqueducts to the scala tympani and round window. The cochlear orifice of the aqueduct is a mere one to two millimetres from the round window membrane in the scala tympani.

The cochlear aqueduct runs from the scala tympani to the anterior part of the bony recess for the jugular bulb. It is 12·9 mm. long and 0·14 mm. at its narrowest

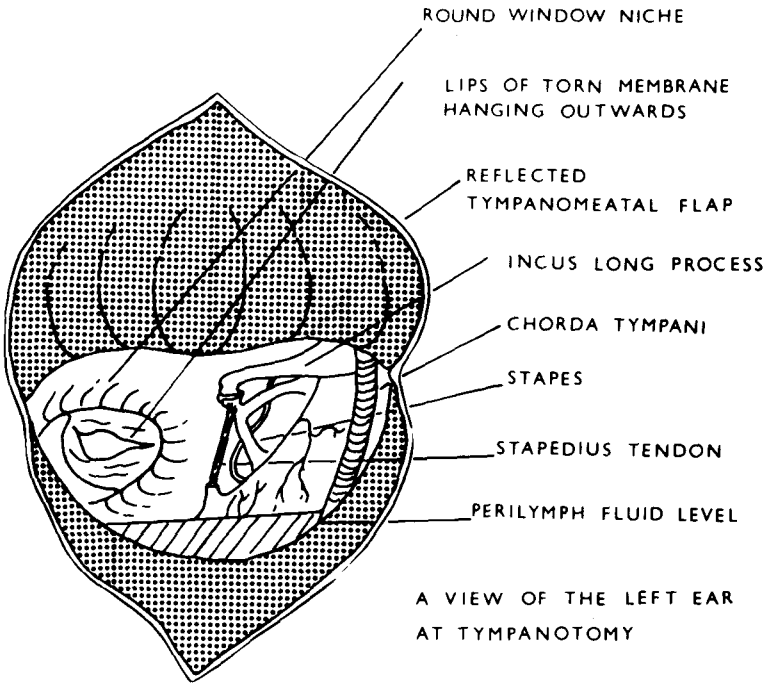


FIG. 2

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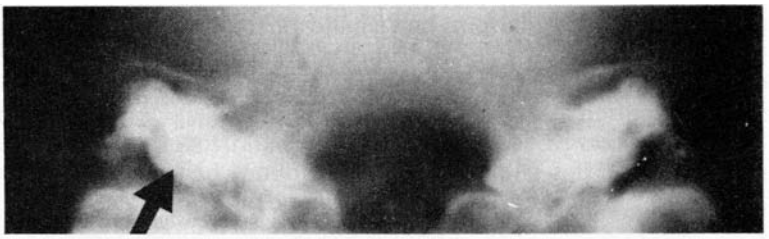


FIG. 3

point (average values for 80 specimens). It lies in a plane parallel to the internal auditory meatus and it can be seen in A–P tomograms (Fig. 3). In 60 per cent of cases the aqueduct may be visualized in its entire length (Rask-Anderson *et al.*, 1977).

The hypothesis of abnormal patency was tested in three of the patients in the group (J.W., A.C., P.W.). The 11·5 mm. cut shows the aqueduct (arrowed) best. Although this pair of aqueducts could not be seen in their entire length, they were considered radiologically patent and normal in appearance. Pathological dilation was not in evidence, and the 'infantile' short wide duct could not be demonstrated in any of the ears examined radiographically.

Fig. 3 shows a normal cochlear aqueduct (arrowed).

2. *Head injury*

This causes skull deformation and a rise in pressure of the incompressible intracranial fluids. These fluids rupture their containing membranes at the weakest point when these are stressed beyond their elastic limit.

Three possibilities exist. Deformation of the petrous temporal bone may force labyrinthine fluids to escape from the windows or displacement of CSF may occur along one of Goodhill's 'explosive' routes (Table I). The fact that the vault of the skull is more accessible to injury and deformable than the base lends weight to the second hypothesis. However, in one patient (A.G.) window rupture was associated with a facial palsy which had not recovered after seven months. The third possibility is that the inertia of the ossicles with a sudden acceleration of the head forces them to move beyond their normal range and tear the labyrithnic membranes.

3. *A blow on the ear*

The two patients in this group had no evidence of a tympanic membrane perforation when they were first examined three and seven weeks after injury.

A pressure wave travelling along the external auditory meatus passes energy to the ossicular chain, the excursion of which is beyond the normal range.

On reaching the oval window, the energy wave passes up the scala vestibuli and down the scala tympani via the helicotrema.

Deformation of the scala media and the organ of Corti is certainly beyond the normal range.

Reaching the round window, the energy may be dissipated in rupture of the membrane as in our patients or in squeezing perilymph along the cochlear aqueduct or endolymph into the sac.

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In theory, endolymph may be displaced into the utricle, saccule, semi-circular canals, vestibular aqueduct or endolymphatic sac.

Whether the round window membrane is forced out by middle-ear pressure, or in by a rebounding ossicular chain on the first trough of the pressure wave, has been debated many times.

In our case (T.A.) minute observation of the torn membrane showed that the 'lips' of the tear were hanging outwards (see sketch), thus favouring the theory of a blow-out rupture.

4. *Barotrauma*

Two sources of energy are at work here:

(i) Middle-ear gases obeying Boyle's law.
(ii) Conscious adjustment of the middle-ear space volume and pressure by 'ear clearing'.

(i) *Boyle's law damage*

On descent, the middle-ear space volume falls with increasing external pressure. All the moving parts of the middle and internal ear may be taken beyond their working range. A conscious readjustment of pressure by the diver is evoked by a sensation of discomfort or pain or the habits of his training. On ascent, the middle-ear volume rises and when a pressure of 200 mm. (20 Kpa.) of water greater than ambient occurs, the Eustachian tube opens passively to vent excess gas. Barotrauma on ascent is very rare (Edmonds *et al.*, 1976). Case 4, however, is an example.

(ii) *Ear clearing*

This alone may cause damage to middle or inner ear structures. Divers are commonly taught the Valsalva method of forced expiration with pinched nostrils but with an open glottis. They frequently do this in the head down position. This is relevant, because passive hydrostatic filling of the venous plexus in the Eustachian tube mucosa narrows the tube in this position, a fact of which few diving instructors are aware (Rundcrantz, 1969).

Passenger aircraft cabins are customarily pressurized to 8,000 or 10,000 feet to limit the range of pressure changes and the hypoxia which would be fatal at the height of 30–40,000 feet where most jet airliners fly for speed and fuel economy. Despite pressurization of the cabin, the pressure at 10,000 feet is about 0·6 bars or 60 per cent of the atmospheric pressure at sea level. Thus an air traveller descending from 10,000 feet (0·6 bars approximately) to sea level (1 bar) suffers an increase in pressure equivalent to a diver descending from the surface (1 bar) to 13 feet (1·4 bars). The rate of change is an important factor, for the change occurs in 10 minutes or longer in air flights, but the diver has only 10 to 20 seconds to accomplish this change.

The sensation of tension in the tympanic membrane, ossicular ligaments or joint receptors lies in the range 100–200 mm. H_2O (1–2 Kpa.) but 2,000 or 3,000 mm. (20–30 Kpa.) may be tolerated before pain is felt. Thus with a change in pressure such as shown above, the number of separate conscious ear clearing manoeuvres by a passenger during an aeroplane descent or in a comparable shallow dive may vary between 1 and 10. It is not surprising that rupture is reported more frequently from diving experiences than from flying.

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A diver may find one ear clears readily but the second requires greater lung pressure. The correct procedure is to ascend ten feet and try again, preferably in an upright posture. Frequently, however, the diver simply increases lung pressure as high as is required or he is able. In a fit, adult male, this may be considerable. The author's (RFGs) personal best is 173 cm. of water (17·3 Kpa.).

The pressure in the easily cleared non-troublesome ear may be sufficient to rupture the round window or tympanic membrane and this has been documented by Edmonds *et al.* (1976).

Harker *et al.* (1974) studied the round window in anaesthetized cats following a sudden artificial rise in CSF pressure. After a delay of ½ to 1½ seconds, the membranes bulged and ruptured in many cases. The lowest recorded pressure to produce a rupture was 1,280 mm. Hg. or 163·2 cm. of water (16·3 Kpa.). Compare this with the pressure developed in the author's chest (above). In the head down position adopted by many divers there would be an even greater pressure transmitted to the intracranial contents.

(iii) *A combination of both* (i) *and* (ii)

An unanticipated descent by a diver, combined with difficulty in clearing a 'sticky ear', is postulated as the association of events which is most likely to swing the moving parts of the middle and inner ears suddenly beyond their working range (Strauss, 1976).

Decompression sickness with nitrogen bubble emboli in the cochlear artery or labyrinthine fluids may cause sudden loss of hearing or balance, and other signs of decompression sickness may be absent. The risks of decompression sickness may be calculated to an accuracy of two per cent by consulting diving depth/duration tables and these will virtually rule out confusion if a round window rupture is suspected.

Recompression, preferably within an hour of onset, is the treatment for symptoms and signs of inner ear damage in divers or pressure chamber workers who have disregarded or abused the decompression tables (Farmer, 1977).

Recompression is strongly contraindicated where diving depths and durations have been within the limits set out in the tables, as the succession of 'ear clearing' manoeuvres required in the chamber is likely to produce further cochlear damage in the presence of 'RW rupture'.

In the rare event of recompression treatment being urgently required for the treatment of air emboli from a damaged lung, in the absence of the possibility of emboli of accumulated nitrogen, the treatment of circulatory embarrassment or neurological deficit must take precedence over cochlear damage if a labyrinthine fistula is suspected.

Electrocochleography

One patient (A.C.) underwent electrocochleography both before and after RW rupture repair. These traces are shown in Fig. 4.

In all, four of the eight patients had electrocochleography and the results are summarized. Unfortunately, no trace is available both before and after repair in a case where there was a substantial gain in hearing.

It is probably naive to suppose that round or oval window membrane rupture when observed is the only lesion which accompanies mechanical stress on the cochlea. Internal membrane rupture may occur without external leaks.

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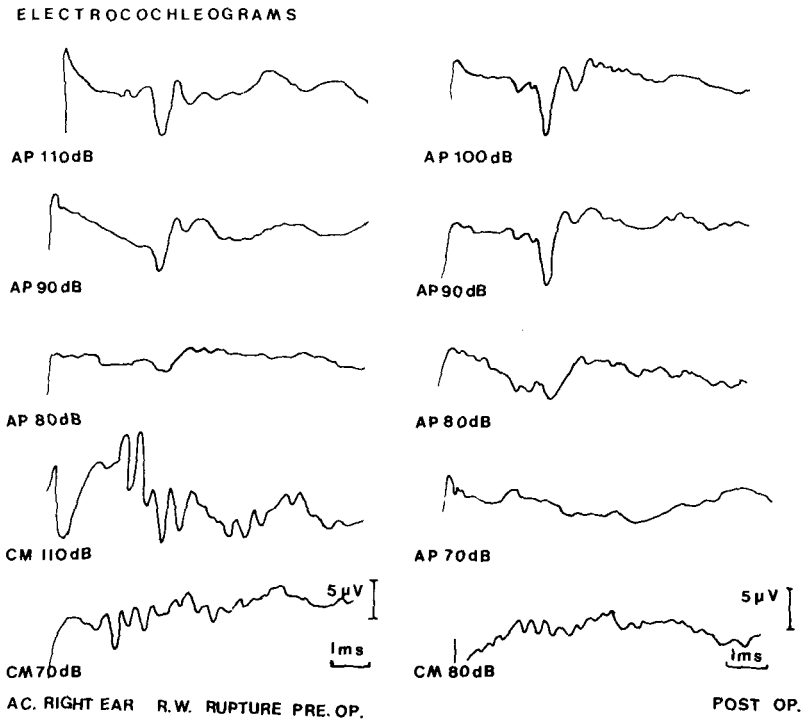


FIG. 4

Other damage must be considered:

1. Detachment of tectorial membrane from outer hair cells.
2. Mechanical damage to cilia.
3. Mechanical damage to the bodies of hair and supporting cells.
4. Microscopical haemorrhages into organ of Corti.
5. Dendrite and dendrite-hair cell junction rupture.
6. Rupture of Reissner's membrane, with loss of endo-cochlear potential.

This last possibility would be expected to produce a reduction in the amplitude of the cochlear microphonic, with reduction in the amplitude of the action potential.

Fig. 4 shows three features typical of the cases tested so far in this way. The compound action potential (AP) is small and fades rapidly at a sound pressure level comparable to the pure tone threshold. There is no change in the trace 4 weeks after RW rupture repair. The cochlear microphonic (CM) is detectable 10 db. lower than the AP threshold. This finding was true for three out of four cases tested.

In summary, electrocochleography has been unhelpful to date, but if a feature of the response can be found which indicates whether the cochlea is merely leaking perilymph or has suffered the internal damage listed above, real progress in the management of vestibulo-cochlear trauma could be made. Careful

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measurement of the summating potential (not done here) might provide the answer as this is frequently associated with reversible cochlear damage, and would therefore be an optimistic prognostic feature.

Conclusions

RW rupture is more common than expected, has at least four aetiologies and may be associated with a congenital variation in round window attitude and cochlear aqueduct patency. Perilymph leaks may be multiple in that group of patients suffering head trauma. The prognosis is poor but probably better with early diagnosis and treatment. Table IV shows hearing gain related to delay in

TABLE IV

HEARING GAIN RELATED TO DELAY IN DIAGNOSIS

Excellent	(i) T.D.	Seven days
	(ii) L.F.	Four weeks
Moderate	(i) J.W.	Seven days
	(ii) A.G.	Ten days
Significant	(i) A.C.	Seven weeks
Nil	(i) P.W.	Seven weeks
	(ii) C.W.	Four weeks
	(iii) T.A.	Eight weeks

diagnosis. Electrocochleography and tomography of the cochlear aqueduct have not been helpful. The diagnosis should be made on pure-tone audiometry and clinical history.

It is remarkable how many divers have been found amongst the sufferers, not only in the water but even when exerting themselves on land. Perhaps repeated pressure changes predispose to subsequent membrane failure.

Divers should avoid forceful Valsalva manoeuvres, especially in the head down position. Unless a labyrinthine fistula can be excluded, the treatment of the 'bends' or decompression sickness by recompression, where the only symptoms are referable to the cochlea and labyrinth, may make the hearing and balance worse.

It is unfortunate that among those cases of RW rupture investigated by transtympanic electrocochleography, there is not an example where hearing markedly improved after surgery. The differences between the pre-operative and post-operative electrocochleograms would be of the greatest interest.

Acknowledgements

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REFERENCES

EDMONDS, C., LOWRY, C., and PENNEFATHER, J. (1976) *Diving and Subaquatic Medicine.* Diving Medical Centre, Australia.

https://doi.org/10.1017/S0022215100090563 Published online by Cambridge University Press

FARMER, J. C. (1977) *Annals of Otology, Rhinology and Laryngology,* **86,** (1 Pt. 3, Supplement 36) 1.
FEE, G. A. (1968) *Archives of Otolaryngology,* **88,** 43.
FRASER, J. G., and HARBOROW, P. C. (1975). *Journal of Laryngology and Otology,* **89,** 1.
FREEMAN, P., and EDMONDS, C. (1972) *Archives of Otolaryngology,* **95,** 556.
GIBSON, W. (1977) *Essentials of Clinical Electric Response Audiometry,* Churchill Livingston, London.
GOODHILL, V. (1971) *Laryngoscope,* **18,** 1462.
HARKER, L., NORANTE, J., and RYU, J. (1974) *Transactions of the American Academy of Ophthalmology and Otolaryngology,* **78,** 448.
MCNICOLL, W. D. (1978) *Journal of Laryngology and Otology,* **92,** 897.
PULLEN, F. W. (1972) *Transactions of the American Academy of Ophthalmology and Otolaryngology,* **76,** 1444.
RASK-ANDERSON, H., STAHLE, J., and WILLBRANCH, M. (1977) *Annals of Otology, Rhinology and Laryngology,* **86,** (5 Pt. 2 Supplement 42) 1.
RUNDCRANTZ, H. (1969) *Acta Otolaryngologica* (Stockholm), **70,** 351.
STRAUSS, R. H. (ED.) (1976) *Diving Medicine,* Grune and Strattan, New York.
STROUD, M. H., and CALCATERRA, T. C. (1970) *Laryngoscope,* **80,** 479.

https://doi.org/10.1017/S0022215100090563 Published online by Cambridge University Press

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