THE CAUSATION OF PERILYMPH FISTULAE IN DIVERS

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On 23 December 1979 a 23 year old man went swimming in a lake. He had no snorkel or SCUBA equipment. He duck-dived to the bottom of the lake (10 feet - 3 metres). He stayed on the bottom by holding on to a rock. Suddenly he noticed a low pitched tinnitus in his left ear. He was not trying to Valsalva at the time. When he left the water the tinnitus disappeared.

Soon after this he had attacks of left sided tinnitus whenever he stooped down or turned his head to the right. On 28 December 1979 he developed, in addition, attacks of vertigo lasting a few minutes whenever he stooped down. At no stage did he notice any hearing loss.

He attended the casualty department at the Royal Victorian Eye and Ear Hospital on 28 February 1980 complaining of postural tinnitus and postural vertigo for 2 months. On clinical examination he had a hearing deficit on the left side. An audiogram revealed a 25 Db sensorineural hearing loss at 400 Hz. Otherwise he was normal, and in particular was fistula negative and Romberg negative.

Thirteen days later (12 March 1980) his audiogram was unchanged when he was operated upon. A perilymph fistula was discovered at the anterior-superior margin of the oval window. The surrounding musoca was scarified and the fistula plugged with ear lobe fat.

Post-operatively the positional vertigo disappeared. The original low pitched positional tinnitus diminished.

His first post-operative audiogram (17 March 1980) showed a slightly increased sensorineural deafness at 3000 and 4000 Hz. At this examination his ear was noted to be bleeding. A repeat audiogram the next day showed a large air-bone gap, and increased sensorineural deafness. Until this episode of bleeding from his ear there was little change in his hearing. Presumably the haemorrhage dislodged the fat graft and inflicted damage on his inner ear.

Follow up audiograms have shown a steady reduction in the air bone gap (conductive element) and some improvement in the sensorineural deafness.

DISCUSSION

a. Diagnosis.

This case confirms the points made by Molvaer.[1] Especially it is necessary to take a careful history when the patient presents with aural symptoms such as tinnitus. Tinnitus is a common presenting complaint of perilymph fistula. Vertigo often follows by several days and hearing loss is often understated.

b. Mechanism of Injury

In order to rupture a membrane the pressure must be markedly different on the two sides. A raised pressure in the fluid in the inner ear with a normal middle ear air pressure fulfils this condition. This pressure rise is achieved by:

a. raising the CSF pressure and transmitting the pressure rise to the perilymph.
b. by compressing the CSF (so raising its pressure)by implosion of the stapes into the oval window

In either case there can now occur a true explosive fistula, one due to a higher inside pressure.

The likely explanation for the perilymph fistulae of breathhold divers is that there is excessive inward movement of the stapes due to excessive inward movement of the ear drum as a result of the rapid rise in ambient pressures in a diver who does not clear his ears (equalise middle ear pressures with ambient) as he descends. This could either rupture the membrane round the foot plate or stress the round window beyond its elastic limits. In either case creating a fistula. In some divers the ear-drum ruptures before damage is done to the inner ear windows.

Goodhill[2,3] postulates explosive and implosive routes for the causation of perilymph fistulae. The explosive force being CSF transfer through the cochlear aqueduct, and perhaps through the internal auditory meatus and lamina cribrosa. This explains the perilymph fistulae occurring when effort and hypertension cause a CSF pressure rise. His implosive route (which includes Valsalva manoeuvres) is less clear as he implies that the fistulae are due to the sudden rise in middle ear pressure, without specifying the mechanical effects in the middle ear which damage the windows.

A membrane supported on one side by fluid is unlikely to be deformed by any rise in air pressure on its outer side since water is virtually incompressible at physiological pressures. Any rise in middle ear pressure must press equally on oval and round windows unless the ear drum moves in response to the rise in pressure. Goodhill's diagram 37-8 3 showing implosive routes is mechanically wrong. Normal stapes movement produces normal round window movement. Excessive compression of the perilymph by stapes movement at the oval window will result in a pressure rise at the round window and at the perilymph - CSF transfer points. If the cochlear aqueduct, lamina cribrosa and internal auditory meatus can transfer some fluid away from the inner ear the pressure rise in the inner ear will remain below that required to deform the round window beyond its limits. However the mechanical movement of the stapes may overstress the footplate membrane, so rupturing it and relieving the pressure in the inner ear before the round window membrane fails.

It has been postulated[4] that with a forceful Valsalva manoeuvre occurring suddenly the ear-drum is blown outwards pulling the ossicles suddenly outwards. The stapes is then exerting a negative pressure on the inner ear and as a result the round window is sucked in beyond its elastic limits. Alternatively the oval window might crack first so causing an oval window fistula. It is difficult to imagine that there would be enough movement of the ear-drum, which

has been forced in by unopposed water pressure, in a diver under water for this to happen. It seems quite possible in air and could explain the hearing loss associated with an oval window fistula reported by Friedman and Sasaki[5] during positive pressure resuscitation. However, this mechanism is not implosive. The only implosive injury is that of the diver who does not clear his ears (equalise middle ear pressures with ambient) as he descends rapidly.

The reason now advanced for the Valsalva induced fistulae in divers is that they are explosive due to the accompanying CSF pressure rise.

A further mechanism occurs in SCUBA divers during the ascent phase of the dive. A case was described by Mannerheim[6] where the diver sustained both a perilymph fistula and a ruptured ear drum during ascent. Not satisfied with a perfect recovery of his hearing following gelfoam packing of his round window niche he repeated the performance some months later. During the second operation photographs were taken showing a steady drip through a split in the round window membrane. Again packing with gelfoam resulted in a restoration of normal hearing.

The likely mechanism on both occasions was that he failed to clear his ears adequately on descent. The pressure imbalance resulted in transudation of fluid,[7] which probably clotted and blocked the Eustachian tube. Ascent resulted in pain, as reduction of ambient pressure resulted in the middle ear having a higher pressure than its surroundings. As a result the ear-drum was forced outwards. Depending on the relative strengths of the membranes the drum or one of the windows ruptures first. If a window goes first the drum will follow. However if the drum goes first the inner ear membranes are protected by the return of the stapes to its normal position.

Goodhill2,3 reviewed 76 cases of presumed perilymph fistulae including 17 who were not explored so cannot be confirmed as having had a fistula. In 59 explorations, 47 fistulae were found. 24 of the patients had fistulae of the oval window alone, 19 had fistulae both of oval and round windows and only 4 had fistulae of the round window alone. He did not give a breakdown into implosive and explosive aetiologies, nor did he mention how many suffered their damage while diving. The left ear was affected twice as often as the right.

c. Operative Technique.

Goodhill recommends the use of tragal perichondium. The fistula was patched with fat in our case. The initial results were good but the events following the bleed in the 5th post operative day suggest that the fistula was still unhealed at that time. In our experience two cases where the leaks were patched with crushed gelfoam had prompt cessation of the leak, and steady improvement in hearing from the time of the operation. As these fistulae are linear tears (which heal from side to side once the edges are approximated) the important thing is to provide a firm splint to hold the edges together and so allow healing by stopping the leak.

CLASSIFICATION

In view of the above we offer a more physiological classification.

Type 1

Due to raised CSF pressure transmitted to the inner ear (Goodhill's Explosive). Acting by bulging the window membranes beyond their elastic limits.

Type 2

Due to an excessive outward movement of the eardrum and ossicles creating a negative pressure in the inner ear (wrongly called implosive by Goodhill). Found in SCUBA divers during ascent and with positive pressure ventilation.

Type 3

Due to an excessive inward excursion of the ear drum and ossicles creating a positive pressure in the inner ear. (Correctly called implosive by Goodhill). This mechanism requires:

a. a rapidly rising ambient pressure,

b. failure to equalise the middle ear pressure with ambient,

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