

Invited editorial

Diving and inner ear damage

Carl Edmonds

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The most recent and comprehensive review of the history of this subject was made by Shannon Hunter and Joe Farmer, of Duke University, Durham, North Carolina.¹ Most of my references are in that document, and I will restrict myself to a brief overview and some recent observations.

Those who do not know the past are doomed to repeat it. In the 19th century A H Smith described 'caisson disease', affecting the ear. Alt and Heller demonstrated inner ear injury during compression and decompression and identified the cause in both humans and animals. Heller described 24 cases of a Ménière-like syndrome, and noted that these cases had undergone very long exposures in caissons. Vail, in 1929, eloquently explained the pathophysiology of the effects of both barotrauma and decompression sickness (DCS) on the inner ear.

Then, somehow, all this was forgotten. World War Two saw an increase in diving, and men from the artillery became divers. Hearing loss was attributable to noise exposure and gunfire, and otologists dismissed the possibility of diving/caisson-induced inner ear damage. In the diving world until the 1970s, the consensus was that diving only induced transitory middle ear damage, called ear squeeze or reverse squeeze.

Rubenstein and Summitt, of the US Navy Experimental Diving Unit, described 10 cases of inner ear decompression sickness (IEDCS) caused mainly by very deep diving.² All presented with severe vertigo, some with tinnitus and sensorineural deafness. Buhlmann and Gehring, from Zurich, described another 12 cases, mainly the result of deep heliox dives.³ Farmer and his colleagues from Duke University added another 13 cases, and the concept of IEDCS was established and defined.⁴

Few of these cases resulted from recreational dives. They tended to be very deep air or heliox dives, precipitated by gas switching to air, and free from other manifestations of DCS. These observations were substantiated when Lambertsen and Idecuola, in 1975, observed vestibular lesions while breathing a heavy, slow-moving gas (air, neon) in a fast-moving gas environment (helium), this counter-diffusion of gases occurring at a constant depth.⁵

Canadian researchers demonstrated the bubble production that characterises DCS within the first few hours and its

conversion into haemorrhages and exudates in the inner ear.⁶ Bubble enucleation in the osteoplastic cell cavities around the inner ear increases local pressure and may disrupt into the perilymph spaces. This pathology is converted, after a month or so, into fibrosis and new bone formation around the permanently damaged inner ear.

This explains why recompression therapy must be instituted rapidly, within the first three to six hours. If the diver has surfaced, a conventional long US Navy oxygen table is employed. If IEDCS develops during decompression, reversion to the original gas mixture, immediate descent to incident depth plus 3 Ata (303 kPa), and then oxygen supplementation, is indicated.

The above observations all seem to describe discrete entities with little confusion regarding diagnosis or symptoms. There have been occasional isolated cases of air divers and caisson workers, comprehensively documented and investigated, developing DCS including inner ear symptoms, such as those described by Reisman et al.⁷ Excessive exposure (duration) was customary. Unfortunately other series of alleged IEDCS cases have less verifiable diagnoses.⁸ This also applies to the current presentation, in which Wong and Walker highlight the difficulty in differential diagnosis.⁹

Such difficulties do not plague the diagnosis of inner ear damage from barotrauma (IEBt). This terminology was introduced by Freeman and Edmonds, when they documented and demonstrated these injuries by pre- and post-incident audiograms.^{10,11} They showed that permanent hearing loss can follow barotrauma and that the pathology included round-window fistulae (RWF). The first two corrective operations for RWF were performed, very successfully, in Sydney in 1971. The Australian workers not only demonstrated sensorineural hearing loss, but also vestibular lesions, in the first text on diving otology.¹² Because the cases were clearly diagnosable, there was little contention when the findings disputed the conventional belief that air diving had no permanent audiological effects. That original work has not only been verified by many others, but is now complemented by a recent series of 50 similar cases.¹³

Sensorineural hearing loss, possibly with loud tinnitus and/or dysacusis, should always imply a possible diagnosis of IEBt. Pure tone audiometry (PTA) is essential in assessing diagnosis and severity. Vestibular symptoms may be of any severity, and sometimes the electronystagmogram (ENG) may demonstrate damage even without symptoms.

IEBt is usually associated with middle ear barotrauma (MEBt), and forceful attempts at middle ear equalisation (ME=), usually by a Valsalva manoeuvre. This increases pressure within the intracranial fluid and through its cochlear duct to the perilymph. These explosive pressures distort the inner ear membranes, one of which is the round

window. The latter may tear, producing a fistula and leaking perilymph, or stretch, allowing effusion of perilymph into the middle ear or air into the inner ear. Haemorrhages within the inner ear are likely.¹

The explosive pressure from an attempted Valsalva is aggravated by the under-pressure in the middle ear (MEBt of descent). Implosive pressures can develop with over-pressure within the middle ear during ascent and with movement of the ossicular chain during sudden successful Valsalvas.

Oval-window fistula is less common than RWF, except in divers with otosclerosis or previous middle ear surgery. Thus the term 'labyrinthine-window fistula' is more accurate. Both a vertical orientation of the round window, and involvement of the fissure of Hurley, have been associated with RWF.

The pathophysiology guides the treatment of IEBt. The patient is advised to cease all activities that increase intracranial pressure (ME=, lifting, exertion, nose blowing or sneezing, coughing, sexual activity, straining at stool, etc.) He is kept at bed rest with the head elevated. PTA performed daily will monitor progress, as the membranes heal and haemorrhages settle, but if there is either severe or progressive hearing loss, exploration is warranted. Vasodilators and anticoagulants, such as nicotinamide and aspirin, are contra-indicated. Surgery is required less often now that careful attention is given to conservative management.

Improvement or even cure of the sensorineural deafness is possible in the first few weeks. The vestibular symptoms will diminish (unless the perilymph leak continues) over the next few weeks or months as cerebral inhibition of vestibular function develops, or if the vestibular damage improves. ENG's are invaluable in diagnosis, localisation and assessing outcome. They can be performed in recompression chambers.

Differential diagnosis is difficult if the dive profile does not clearly indicate either MEBt or DCS (vestibular or cerebellar). A comprehensive inquiry is needed to exclude MEBt. Other symptoms of DCS may be of value. The simple statement that the diver had 'no trouble with ME=' is inadequate, and a delay between dive and onset of symptoms does not discriminate between diagnoses. In the isolated cases of air divers and caisson workers reported by Reissman et al, excessive exposure (duration) was customary.⁷ Recent cases have been postulated as not requiring such exposure, the inner ear lesion being produced by DCS gas emboli passing through a patent foramen ovale.

Oxygen recompression, although theoretically hazardous with IEBt, has been employed and even recommended as a valid treatment for sudden hearing loss.¹⁴⁻¹⁶

If there is permanent damage from IEBt, most recommend that diving, free or with equipment, cease, although this is contentious. Prophylactic advice should be given regarding ME= with aviation exposure.

Australasians have no reason to be modest regarding their contribution to diving otology. Australian workers have:

- compiled prospective documentation on MEBt (Bayliss, Edmonds, Lehm and Bennett);
- discovered and treated RWF;
- demonstrated and defined the causes of hearing loss and vertigo from diving (Edmonds, Thomas, Tonkin, Freeman, Blackwood);
- described orbital barotrauma¹⁷ and the only two large series of sinus barotraumas (Fagan, McKenzie, Edmonds);^{18, 19}
- produced the first two texts on diving otology, *Otological Aspects of Diving*, in 1973;¹² and, in New Zealand, Noel Roydhouse's *Scuba Diving and the Ear Nose and Throat* in 1975;²⁰
- produced the latest clinical reviews available in diving medical texts;^{1, 21}
- and currently our scientists are modelling the pathophysiology of IEDCS (Doolette, Mitchell).²²

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