6. A DEAF DIVER

Introduction

In 1969 a diver developed a total sensorineural deafness following a compression in a chamber. The aetiological significance of the compression was not clear, as the deafness was noted only after a time delay of a day or more. The depth and duration of the recompression chamber ‘dive’ was such that decompression sickness was not considered a possible cause. This case, and others subsequently to be described, led the author and his colleagues into an investigation into the possible effects of diving on auditory acuity.

Summary of the Literature

Many otologists have interested themselves in the auditory damage which can occur under hyperbaric conditions. Boot (1913) coined the phrase “caisson workers’ deafness” to describe the auditory loss which may be acute of chronic, temporary or permanent. He state that the most characteristic symptom was a loss of a considerable portion of the upper range of hearing. Unfortunately there was no significant discussion on the differential diagnosis of this disorder, only a comment on its occurrence. It was therefore basically a report of historical interest only, proposing an occupational disease. Deafness associated with diving has been reported frequently in the literature, and at least two aetiologies have been demonstrated. A noise induced deafness is not unexpected, and Summitt and Reimers (1971) have indicated the extreme levels likely to be experienced in helmet divers and recompression chambers. In these cases one anticipates either temporary or permanent threshold shifts of the sensorineural type, consistent with noise induced deafness. Decompression sickness has been long known as a cause of inner ear damage, and Harris (1969) reported cases of deafness due to decompression sickness, some of which responded to recompression therapy.

A great deal of the literature was not so clear in its implications. Shilling and Everley (1942) demonstrated that permanent loss of auditory acuity of divers is little greater than would be expected of others of their age. They conceded a greater hearing loss in a miscellaneous group of divers who were also subjected to noise factors. Haines and Harris (1946) highlighted their seemingly contradictory statements on the effects of pressure changes with diving on auditory acuity. They pointed out that some authors claimed a high tone loss, others a low tone loss, some stated that the deafness may be severe and permanent, and others that the auditory acuity was regained within a matter of hours. They also stressed that an

audiogram demonstrating impaired acuity did not necessarily reflect a causal relationship with the diving conditions, unless a previous audiogram had been performed. They stressed the common effects of noise, gunfire, blast, disease, etc. They discredited previous work on these grounds, and performed their own prospective study. They stated that almost no serious or significant effect on acuity was found unless the middle ear was filled with blood unmixed with air, and that deafness was a result more of dampening of the ossicles than of pathological changes. They demonstrated minimal changes both in asymptomatic subjects and those suffering from grades of middle ear barotrauma. Taylor (1959) described one case of permanent sensorineural deafness presumably associated with diving. Unfortunately the absence of pre- and post-incident audiograms for comparison left the conclusions open to the doubts expressed by Haines and Harris years previously. Rawlings (1959) considered the possibility of insidious conductive deafness occurring in naval divers following repeated barotraumatic incidents. Four cases of permanent hearing loss with tinnitus were cited, however, these were refuted by Coles and Knight (1961). The latter authors conducted a small survey on divers and concluded that sensorineural deafness in this group could be explained on the basis of previous exposure to loud noise, particularly gunfire. They reported that a review of the literature up till 1960 revealed single cases of sensorineural deafness, but that pre-incident audiograms were not available to support the contention that sensorineural deafness could result from barotrauma.

During the following decade ample evidence was submitted to verify the existence of a specific isolated disease entity resulting in partial or total sensorineural deafness in divers associated with the performance of the Valsalva manoeuvre. McFie (1964) described three cases in which hearing damage appeared to occur due to diving. In one case auditory loss was demonstrated, and was then aggravated following subsequent diving exposures. He also demonstrated involvement of vestibular function. Unfortunately McFie did not give adequate decompression information about two of his cases. Eichel and Landes (1970) also reported two cases of sensorineural hearing loss caused by skin diving, however as these did not have any pre-incident audiograms, they contributed nothing to the controversy, and were thus subjected to the same criticism as had been levied against previous workers.

1970-1972 Research Reports

Edmonds (1970), working at the RAN School of Underwater Medicine, described seven cases of sensorineural deafness, two total and five partial, in divers who experienced difficulty in performing the Valsalva manoeuvre on the side affected, during descent. The major

characteristic of this series was that pre- and post-incident audiograms were available, and there was a clear aetiological relationship noted between excessive force required in performing the Valsalva manoeuvre during descent. The two cases of total deafness developed progressively in the first few days following the diving incident. Most of the cases were associated with tinnitus continuous from the time of the incident, and some had a demonstrable vestibular dysfunction.

The question that arose as to whether these cases represented an isolated disease entity, associated with this particular occupation, or whether they represented the extreme of a whole range of hearing damage associated with diving, ie. was the hearing damage associated with diving and Valsalva manoeuvres. If the first of these possibilities is correct, then one would expect that only certain divers would show any tendency to this form of sensorineural deafness, whereas the vast majority of the divers would remain unaffected. If the second possibility is correct, and an analogy to this is the noise induced sensorineural deafness loss of many other occupations, then a large number of divers who perform the Valsalva manoeuvre, would be affected to a variable degree. In the latter case, some would be expected to be severely affected, just as some noise induced deafness can be severe. To clarify this position Gray and Edmonds (1970) carried out a prospective study to assess the common effects of diving on the auditory acuity. These experiments were conducted to demonstrate the following:

a. The effects of repeated Valsalva manoeuvres on the auditory acuity;
b. the common effects of middle ear barotrauma of descent on auditory acuity;
c. the endurance of any of the changes noted in experiments a. and b.;
d. the auditory acuity of divers without specific otological disorders, compared to a control population.

These results demonstrated that unless one develops middle ear barotrauma there is unlikely to be any appreciable change in hearing acuity due to diving. If middle ear barotrauma does occur then the hearing loss extends through the 500-8000 cps range, and although it is statistically significant, it is quantitatively very small. This hearing loss is reversed over the ensuring one to three weeks. The temporary and mild impairment of hearing associated with aural barotrauma is conductive in type with a possible sensorineural component. It was decided that the exceptional cases of sensorineural deafness following middle ear barotrauma of descent with forceful Valsalva manoeuvres, is likely to be a disease entity per se, and that there is no evidence from the above experiments that such a change is either common or cumulative from many small such

incidents.

The hearing loss which is associated with other aspects of diving eg. decompression sickness, exposure to loud noise (compressors, recompression chamber environments, etc.) and the temporary 5-10 dB loss with perforation of the tympanic membrane, have all to be clearly differentiated from the above discussion on hearing loss associated with forceful Valsalva manoeuvres during the diver's descent.

Once it had been decided that this hearing loss was a specific disease entity affecting only certain susceptible individuals, conjecture was made regarding the possible aetiology. Some cases in the literature had been treated with vasodilators such as nicotinic acid , in the belief that the cause is a vasospasm. Other cases had been attributed to haemorrhage within the internal ear, a pressure wave directly damaging the auditory and/or vestibular end organs, exceptional manifestations of decompression sickness, a manifestation of middle ear barotrauma of descent, etc.

Freeman and Edmonds (1972) postulated the sequence of events as follows. Divers during descent require to equalise the pressures with in the middle ear cavity. Any delay in this, eg. due to difficulty in performing this Valsalva manoeuvre, will result in an inward movement of the tympanic membrane and the handle of the malleus, which in turn moves the foot plate of the stapes and the oval window. The diver notes pain as the middle ear volume diminishes, and then attempts a more forceful Valsalva manoeuvre. In the event of this being successful there is a sudden and dramatic increase in both gas pressure and gas volume of the middle ear, with a forceful movement of the tympanic membrane and handle of the malleus outwards, again moving the foot of the stapes and the round window. It was postulated that the pressure wave so resulting would cause the hearing and vestibular damage, although the precise nature of this damage was not elaborated.

Pullen (1972) is preparing for presentation as series of four cases of sensorineural deafness which were explored surgically, and in three of these a rupture of the round window was noted. It is of interest to note that one of these cases did occur in a diver, and Freeman, being aware of these developments, recommended that future cases of sensorineural deafness apparently related to the forceful Valsalva manoeuvres with diving, should be explored to exclude this condition which is able to be improved with reconstructive surgery. Preparations were made in the expectation that such a case would occur, in both Sydney and Melbourne, and during the June of 1972 such a case did present. A commando had been diving in 30 feet of water, and experienced considerable pain and difficulty in clearing both ears, but had continued to dive forcefully performing Valsalva manoeuvres. He noted tinnitus, and also experienced ear pain and vertigo during ascent. The tympanic membranes showed the effects of the barotrauma, and the diver progressively became more deaf, with a sensorineural pattern, over the subsequent few days. As both ears were affected, it was considered necessary that explorative surgery be performed and this was carried out, following routine audiometric and vestibular function assessments, by Dr John Tonkin. Damage to

the round window was witnessed, together with an outpouring of fluid into the middle ear from this opening. The round window was packed and subsequent audiograms demonstrated a very considerable improvement in hearing. A similar operative procedure was performed some days later by Dr John Tonkin and Dr Peter Freeman, on the patient's other ear.

DISCUSSION

The implications of the literature review, leading on to the experiments performed and finally the surgical verification of the disease entity require understanding by all medical officers involved with divers. It has become axiomatic that audiograms must be performed on all divers prior to exposing them to hyperbaric conditions. Without these pre-incident audiograms assessment of hearing damage would be most difficult, especially as it predominantly involves the high range frequencies in the less serious cases. It becomes even more important to stress that forceful Valsalva manoeuvres not be performed if there is any difficulty with descent, as an alternative the diver should ascent and discontinue diving until the Valsalva manoeuvres are more easily performed. It is necessary that all hearing abnormalities be referred to personnel experienced in this field, so that confusion does not occur with other disorders associated with diving eg. otitis media, middle ear barotrauma of descent, haemotympanum, etc. Liaison between the diving medical officer and an otological specialist experienced in internal ear surgery is essential. The diving medical officer will have experience with the differential diagnosis, and will also be able to advise on general management eg. he will be aware that any normal air transport of the patient is absolutely contraindicated because of the subatmospheric pressure changes which must occur with this transport. The value of a surgeon experienced in this experimental field is axiomatic. Information from Pullen's cases suggests that a recurrence is possible following activities which may increase intraaural pressures - and divers are very likely to perform routine Valsalva manoeuvres, almost by habit. For this reason careful supervision of the patient post-operatively is advisable.

CONCLUSION

The development of our knowledge of hearing loss in divers has proceeded to such a stage that many of the causes can be clearly diagnosed, and effective treatment instituted. The inclusion of a new cause, and perhaps the most common, is worthy of note and it is proposed that this be named 'perforation of the round window', and replace the less specific terminology of 'internal ear barotrauma'. The importance of reconstructive surgery is stressed.