CASE REPORT

Report of an isolated mid-frequency hearing loss following inner ear barotrauma

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Butler FK Jr, Thalmann ED. Report of an isolated mid-frequency hearing loss following inner ear barotrauma. Undersea Biomed Res 1983; 10(2):131-134.—A case describing an isolated mid-frequency hearing loss as a result of inner ear barotrauma is presented. The onset of symptoms was insidious but progressed to a profound total-range hearing loss in the right ear. This loss resolved rapidly with cessation of diving activity, bed rest, and head elevation, leaving only an isolated 20-dB hearing decrement at 1000 Hz. Since the diver was participating in evaluation of experimental decompression tables, differentiation had to be made between barotrauma and inner ear decompression sickness.

barotrauma
decompression sickness
ear
vestibular function

During large dive series involving many divers who are expected to dive daily, ear barotrauma of all degrees is prevalent. This barotrauma can result in significant hearing loss that may be perceived as minor by the diver. This paper describes such an incident that occurred during a large dive series to evaluate experimental helium-oxygen decompression tables. The clinical management in this case was complicated by the possibility that inner ear decompression sickness caused the symptoms. Differentiation between the two possibilities was important, since the appropriate treatments are quite different.

CLINICAL PRESENTATION

The patient was a 21-year-old male caucasian Navy diver with no previous history of ear problems or significant diving-related inner or middle ear trauma. He was participating in a series of dives at the Navy Experimental Diving Unit designed to ascertain no-decompression limits for a helium-oxygen breathing mixture with a constant oxygen partial pressure of 0.7 ATA. He first reported a medical problem on Thursday, January 21, 1982, stating that on the previous day he had had difficulty clearing his right ear during descent to 60 ft (18 m) for a 20-min chamber indoctrination dive. A sharp, transient pain was noted in the right ear during ascent and the patient began to have mild tinnitus in the right ear shortly after the dive. There
was also a small amount of bloody mucus in the right nostril that was felt to be due to a sinus squeeze. The patient had no previous history of tinnitus, and there was no complaint of hearing loss, vertigo, nausea, vomiting, pain, weakness, or numbness. The tinnitus was described as a "buzzing" and was persistent. Physical examination of the right ear at that time revealed no erythema, perforation, or bulging of the tympanic membrane, and there was no loss of landmarks or evidence of middle ear effusion. There was no ataxia; Romberg's sign was negative; and no hearing loss was detected clinically. No nystagmus was noted. He was felt to have tinnitus secondary to mild middle ear barotrauma and was allowed to participate in his group's dive for that day—60 ft for 130 min. He reported no change in his symptom as a result of this dive. No symptoms of decompression sickness were observed in any of the 10 divers on this dive profile.

On Friday, January 22, the patient made a dive to 160 ft (49 m) for 10 min and had trouble clearing his ears on descent. After the dive, he noted that the tinnitus was louder and felt that there was an element of mild hearing loss as well. Examination of the right tympanic membrane revealed no change from before the dive except for the presence of mild erythema. Again, the other 9 divers noted no symptoms compatible with decompression sickness. The patient was felt to have an exacerbation of his middle ear barotrauma and was suspended from diving the following day (Saturday, January 23). When the patient was seen again on Monday, January 25, he reported that his tinnitus and hearing loss were still present but much improved. He stated that he was able to clear his ears and felt ready to dive. He subsequently made a dive to 60 ft for 130 min and had slight trouble clearing his ears on descent, but no pain. That evening, he noted that the tinnitus and hearing loss in his right ear were much worse. At no time did he have any symptoms referable to his left ear. There was still no vertigo, pain, nausea, vomiting, weakness, nystagmus, or ataxia. Examination on Tuesday, January 26, again revealed only mild erythema of the right tympanic membrane without bulging or loss of landmarks. Once again, none of the other 9 divers had any symptoms of decompression sickness from this dive. A Békésy audiogram performed at this time revealed a diffuse hearing loss in the right ear of 65–75 dB.

The patient was then referred to the Ear, Nose, and Throat Clinic at the Naval Regional Medical Center in Pensacola, FL, where further audiologic testing showed the right ear hearing loss to be of the sensorineural type with "poor" speech discrimination. The left ear was completely normal. A previous audiogram done on January 6, 1982, had shown normal hearing in both ears. The diagnosis of inner ear barotrauma with possible labyrinthine window rupture was considered and the patient was placed at bed rest with his head elevated. He was suspended from any further diving, and serial audiograms were performed. A subsequent Békésy audiogram on January 29 showed improvement in the right ear, with most frequencies now showing a 15-dB loss, 500 Hz showing a 25-dB loss, and 1000 Hz alone with a 45-dB loss. On February 1 continued improvement was noted with 1000-Hz still abnormal, at 35-dB loss, and by February 12 this had decreased to 30 dB. All other frequencies in the right ear were normal. Speech discrimination testing had returned to normal at this time. Further follow-up in June 1982 showed continued improvement with the 1000-Hz hearing loss at 20 dB.

DISCUSSION

The differential diagnosis of tinnitus and hearing loss of the sensorineural type related to diving includes the following: a) inner ear decompression sickness; b) inner ear barotrauma; and c) noise-induced deafness (1, 2). The possibility of gas embolism was also considered but was discounted for lack of supporting clinical evidence such as uncontrolled ascent, altered
state of consciousness, or chest pain. Noise-induced deafness was not a likely factor in light of the normal pre dive series audiogram. In addition, noise levels in the chamber facility used for this dive series cannot be implicated because all dives were done with the divers completely immersed in a 15- by 30-ft wet chamber that reduced compression and decompression noise to insignificant levels. The association of the onset of symptoms with middle ear baro trauma, and the fact that the dive with which onset occurred was a short indoctrination dive and relatively innocuous from a decompression standpoint, led us to favor a diagnosis of inner ear baro trauma. In addition, in this series of more than 300 no-decompression man-dives, 16 cases of decompression sickness were encountered, but none of these cases had symptoms referable to the inner ear, nor was any hearing loss detected clinically on neurological examination. Isolated inner ear decompression sickness has been more frequently noted with deeper diving than the patient experienced, or a change in breathing medium from helium to air during the later stages of decompression, or both (3). Further retrospective evidence that the insult to the inner ear was not related to decompression sickness is provided by the rapid return of function that the patient experienced without the benefit of recompression, which is not the usual course of untreated inner ear decompression sickness (3).

Freeman and Edmonds described 5 cases of sensorineural hearing loss related to inner ear baro trauma (4). The hearing losses described were all of either the high-frequency or total-frequency types. The deficits found in that study were apparently permanent. Goodhill et al. reported a number of cases of sudden deafness that were found to be associated with rupture of the round or oval window (5). These cases were not associated with diving, although a history of physical exertion was often found to be present just prior to the onset of symptoms. This suggests that the injury may be due to an increase in perilymphatic pressure exerting an outward force on the round and oval windows. This force is reproduced by a diver during a forceful Valsalva maneuver on descent as he tries to clear, and the pressure differential between the inner ear and middle ear is increased by the relatively negative pressure in the uncleared middle ear. Although round window rupture need not occur for hearing function to be compromised in inner ear baro trauma (1), Farmer suggests that all cases of sudden sensorineural hearing loss that occur during a dive in which decompression sickness is unlikely to be treated with a regimen of bed rest, head elevation, and avoidance of actions that increase cerebrospinal fluid or middle ear pressures, such as exercise, coughing, noise blowing, straining with defecation, and the performance of the Valsalva maneuver. Exploratory tympanotomy is considered for those patients who demonstrate no improvement after 4 or 5 days of conservative treatment or worsening hearing after 48 h (2, 6).

Few guidelines have been published regarding when, if ever, patients who have suffered significant inner ear baro trauma should be allowed to resume diving. Dr. Joseph Farmer of Duke University (personal communication), a recognized authority on hyperbaric otolaryngology, has offered these criteria:

1. No significant defects remain in the patient’s auditory function. Evaluation should include audiometry with pure tone thresholds and speech discrimination testing. Particularly important is auditory function at the 500-, 1000-, and 2000-Hz ranges, since these are the frequencies most important for speech reception.

2. No significant defects are present in the patient’s vestibular function, as evaluated by electronystagmogram.

3. A thorough ENT evaluation should be done with special attention to factors that might contribute to eustachian tube dysfunction, such as: allergic rhinitis, rebound rhinitis from nasal spray overuse, nasal irritation secondary to smoking, and nasal septal deviation. Any such conditions discovered in this evaluation should be corrected before resumption of diving. Two months is suggested as a minimum period before diving should be resumed.
The patient in this case was allowed to resume diving after his convalescent period. His 20-dB hearing decrement, although in a speech frequency, was confined to this single frequency and produced no decrease in performance on the speech discrimination test. Electronystagmography was not done, since there was a complete lack of any vestibular symptoms. He has since participated in operational diving without any difficulty.

Several factors make this case interesting. One is the insidious onset of symptoms, progressing from a minimal tinnitus to profound hearing loss in the course of several days and several dives. Notable by their absence were other symptoms of inner ear dysfunction such as nystagmus, nausea, or vertigo. The course would appear to be most consistent with middle ear barotrauma at the start complicated by later development of a labyrinthine window rupture that responded well to the regimen of bed rest and head elevation. The second unusual feature is the persistence of a deficit at an isolated mid-range frequency, which is a different injury pattern than the high-frequency hearing losses or total-range hearing loss usually reported with inner ear barotrauma (1, 4).

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baro-traumatisme
maladie de décompression
oreille
fonction vestibulaire

REFERENCES