

INNER EAR DECOMPRESSION SICKNESS IN SPORT COMPRESSED-AIR DIVING

Z. Nachum, A. Shupak, O. Spitzer, Z. Sharoni, Y. Ramon,
A. Abramovich, I. Doweck, C.R. Gordon.

Motion Sickness & Human Performance Lab., Israel Naval
Medical Institute (INMI), IDF Medical Corps, Haifa, Israel.

INTRODUCTION

Inner ear decompression sickness (IEDCS) may result in permanent, severe cochleo-vestibular deficits, unless immediate diagnosis is reached and the correct treatment is commenced early (4). Previously reported IEDCS cases were mostly associated with deep mixed helium diving (1,4), whereas after sport air diving this entity is considered to be rare.

METHODS

Twenty-five divers with sport compressed-air diving-induced IEDCS were referred to the INMI during the last 10 years. They were evaluated, treated, and followed up. All patients were male, average age 35y (22-57y). Evaluation included medical history, physical examination, pneumotoscopy, pure-tone audiometry, electronystagmography and the smooth harmonic acceleration test using a computerized rotatory chair system (NeuroKinetics, Inc., Pittsburgh, PA). Other tests such as CT, MRI or ABR were performed if necessary.

RESULTS

These 25 patients represent 12% of the total number DCS cases and 26% of all Type II DCS treated in that period.

The cause of injury in 19 cases (76%) was violation of the decompression schedule. The mean time from surfacing to appearance of symptoms was 39 min (in 84% symptoms appeared within 1h, in the remainder - within 2h).

Ten divers (40%) had pure vestibular symptoms, 2 (8%) - cochlear, and 13 (52%) - a combination of symptoms. Fourteen (56%) suffered isolated IEDCS, while 11 had additional symptoms of DCS. In 20 divers (80%) injury was to the right ear, while in 5 the left ear was affected.

Eighteen (72%) were treated by US Navy Oxygen Treatment Table 6, two by Comex Table CX-30, two by hyperbaric oxygen, and three divers (one referred only 10 days after the accident, and two misdiagnosed as suffering from inner ear barotrauma) were not treated. Median time from appearance of symptoms to hyperbaric treatment was 4.5h (2-336h). Of the 23 patients with vestibular injury and the 15 with cochlear damage, only 5 (22%) and 5 (33%), respectively, made a full recovery, while the

others remained with residual damage. Follow-up was from 1 week to 4.75y (mean – 12m).

DISCUSSION

Inner ear DCS (IEDCS) follows a rapid ascent, which results in supersaturation of inert gas, producing bubbles within the otic fluids and microvessels. The free gas phase causes blockage of the venous microcirculation of the stria vascularis, spiral ligament and the semicircular canals. These bubbles may also induce activation of leukocytes, endothelial cells, platelets, the coagulation cascade, fibrinolysis and complement systems (7,8).

Animal models have shown hemorrhages and protein exudation in the cochlea. Vestibular damage consisted of irritation of the semicircular canal endosteum with osteoblastic and fibroblastic differentiation, finally leading to fibro-osseous labyrinthitis (7,9).

IEDCS has been reported mainly as resulting from deep oxy-helium diving (1,4), whereas it has seldom been reported after air dives (11,12). This may be due to the rarity of the insult, to misdiagnosis, or to a combination of the two.

When a patient presents with inner ear symptoms after diving, it is important to make the differential diagnosis with inner ear barotrauma. Unlike IEDCS, the pathophysiology of inner ear barotrauma involves the sudden build-up of a high pressure gradient between the cochlear fluid and the middle ear space, resulting in rupture of the round or oval window membranes with perilymphatic leakage into the middle ear (6). The hyperbaric treatment essential for IEDCS would be extremely harmful to the patient with inner ear barotrauma. The treatment of inner ear barotrauma consists of complete bed rest, vestibular tranquilizers, decongestants, and eventually surgical repair of the perilymphatic fistula (2,6). Under no circumstances should these patients be recompressed, as the resulting middle-inner ear pressure gradient can extend the insult in the inner ear. On the other hand, prompt recompression treatment is required for IEDCS if permanent inner ear damage is to be prevented (4).

Much has been written regarding the differential diagnosis between IEDCS and inner ear barotrauma, but the most important factors (4), which also guided us in our cases, include:

- 1) In inner ear barotrauma, the patient will report having had difficulty clearing his ears during pressure changes. In contrast, in IEDCS there will have been no such problems.
- 2) Symptoms of inner ear barotrauma appear during the dive, whereas those of IEDCS appear after the dive.
- 3) IEDCS may be accompanied by other forms of DCS.
- 4) Inner ear barotrauma will often be accompanied by signs of middle ear barotrauma. No such signs will accompany IEDCS.
- 5) In contrast to the improvement observed in IEDCS during hyperbaric treatment, inner ear barotrauma will be aggravated by treatment in the hyperbaric chamber.

The treatment for IEDCS is U.S. Navy treatment table 6 (10). There is general agreement that a shorter time delay to the commencement of treatment will result in a better chance of a complete cure (2,4,5,10).

CONCLUSION

In our experience, IEDCS in sport compressed-air diving is not as rare as previously thought. In divers presenting with suspected inner ear injury, complete otoneurological evaluation process will lead to the correct diagnosis and appropriate treatment.

REFERENCES

1. Buhlmann AA, Gehring H (1976). Inner ear disorders resulting from inadequate decompression – “vertigo bends”. In: Lambertsen CJ, ed. Underwater physiology V. Proceedings of the fifth symposium on underwater physiology. Bethesda, MD: FASEB, 341-7.
2. Caruso VG, Winkelmann PE, Correia MJ, Miltenberger GE, Love JT (1977). Otologic and otoneurologic injuries in divers: clinical studies on nine commercial and two sport divers. *Laryngoscope* 87:508-21.
3. Cyr DG, Harker LA (1993). Vestibular function tests. In: Cummings CW, Fredrickson JM, Harker LA, Krause CJ, Schuller DE, eds. Otolaryngology – head and neck surgery, 2nd ed. Vol. 4: Ear and cranial base. St. Louis: Mosby-Year Book, Inc. Chapter 146: 2652-82.
4. Farmer JC, Thomas WG, Youngblood DG, Bennett PB (1976). Inner ear decompression sickness. *Laryngoscope* 86:1315-27.
5. Flynn ET, Catron PW, Bayne CG (1981). Decompression sickness: clinical manifestations and pathogenesis. In: Diving Medical Officer Student Guide, course A-6A-0010. Naval Technical Training Command, 15-1 – 15-16.
6. Goodhill V, Harris I, Brockman SJ, Hantz O (1973). Sudden deafness and labyrinthine window ruptures. Audio-vestibular observations. *Ann Otol Rhinol Laryngol* 82:2-12.
7. Landolt JP, Money KE, Topliff EDL, et al (1980). Pathophysiology of inner ear dysfunction in the squirrel monkey in rapid decompression. *J Appl Physiol* 49:1070-82.
8. McCormick JG, Philbrick T, Holland W, Harrill JA (1973). Diving induced sensori-neural deafness: prophylactic use of heparin and preliminary histopathology results. *Laryngoscope* 83:1483-501.
9. Money KE, Buckingham IP, Calder IM, et al (1985). Damage to the middle ear and the inner ear in underwater divers. *Undersea Biomed Res* 12:77-84.
10. Moon RE, Sheffield PJ (1997). Guidelines for treatment of decompression illness. *Aviat Space Environ Med* 68: 234-43.
11. Reissman P, Shupak A, Nachum Z, Melamed Y (1990). Inner ear decompression sickness following a shallow scuba dive. *Aviat Space Environ Med* 61:563-6.
12. Shupak A, Doweck I, Greenberg E, et al (1991). Diving-related inner ear injuries. *Laryngoscope* 101:173-9.