VESTIBULAR EFFECTS OF DIVING – A REVIEW

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ABSTRACT

Scuba diving is a popular recreational and professional activity with inherent risks. Complications related to barotrauma and decompression illness can pose significant morbidity to a diver's hearing and balance systems. The common symptoms of otologic dysfunction are ear fullness or pain, hearing loss, tinnitus, and vertigo. Vertigo is one of the most hazardous symptoms to occur during diving. It is frequently accompanied by hearing loss and tinnitus. Vertigo is described in multiple phases of diving. However, many of these reports are not well documented, do not differentiate vertigo from nonvertiginous disequilibrium, or discuss vertigo only as an incidental observation.The purpose of this publication is to review the vestibular effects of diving.

Keywords: *vestibular effects, diving, vertigo, otologic dysfunction*

INTRODUCTION

Divers occasionally suffer vestibular injury due to barotrauma or decompression illness (DCI) (15). Acute cases are usually recognized by a combination of vertigo, nausea and vomiting with a tendency to fall to the injured side and nystagmus to the contralateral side. The loss of peripheral vestibular function may be permanent, but in spite of this, there is often a nearly complete symptomatic recovery after weeks to months due to central vestibular compensation.

AIM

The purpose of this publication is to review the vestibular effects of diving.

MATERIALS AND METHODS

The following research databases were used for the study: MEDLINE, Scopus, EMBASE and PubMed from January 2020 to August 2020, using the following keywords: marine medicine, vestibular system, diving.

RESULTS AND DISCUSSION

Less dramatic but much more common are middle ear barotraumas and short episodes of vertigo during diving. Middle ear barotrauma is the most common medical problem in diving (24). It is characterized by ear pain, usually during descent, conductive hearing loss, and otoscopic signs of barotrauma to the middle ear (haemotympanum) or the tympanic membrane (haemorrhage and/or rupture). The prognosis is usually excellent but repeated barotraumas may cause permanent hearing loss (5).

Barotrauma is a pressure-mediated injury to tissue governed by Boyle's law. As a diver descends and the pressure increases, the volume of the gas compresses. This can result in a relative negative pressure in rigid- or semi-rigid-walled air-containing spaces in the body such as the middle ear or paranasal sinuses. Negative pressure can cause mucosal oedema, haemorrhage and even perforation if the space cannot equalize. On ascent, the volume of gas increases as the ambient pressure decreases. If an air-containing space cannot equalize with the surrounding pressure, the expanding volume of the gas may result in a variety of head and neck pathologies, including middle ear perforation or pneumocephalus from sinus barotrauma (7).

Tinnitus, or spontaneous noise in the ear, is difficult to quantitate because different patients experience different types of noise. Also, the perception of severity and the effect on daily life vary. Tinnitus

can occur with middle ear disease that results in a conductive hearing loss but usually occurs with inner ear or central auditory pathway disease. With the former, tinnitus is thought to represent the sounds of cochlear and intracranial blood flow that are perceived because the conductive hearing loss results in a loss or a decrease of the masking effect of the usual background noise. Patients may describe a rushing, pulsatile sound that can often be timed with the pulse. With inner ear disease, tinnitus is thought to be due to the spontaneous firing of injured but viable auditory neurons or cochlear hair cells. However, this is not well understood: destructive labyrinthectomies or 8th cranial nerve sections in patients with recurrent and disabling vertigo, tinnitus, and nonfunctional hearing due to Meniere disease—which usually relieve the vertigo—have frequently not relieved the tinnitus.

Vertigo while diving is common (Table 1). Edmonds et al. (6) undertook a complete review of the various causes of dizziness in diving. Their classification is basically broken down into those causes of vertigo due to unequal vestibular stimulation, including caloric stimulations, barotrauma, and decompression sickness; and those due to unequal vestibular responses to equal stimuli, such as the result of one vestibular apparatus being more sensitive than the other. Affected persons might have vertigo with caloric stimulation resulting from equal amounts of cold water entering the external ear canals. Also included in this group are divers who experience vertigo resulting from a unilateral hypofunctioning vestibular end-organ in situations in which equal and symmetrical pressure changes occur in the middle ear cavities during ascent and descent.

In addition, this classification includes dizziness noted with nitrogen narcosis; the dizziness, nausea, and tremor described in the high-pressure nervous syndrome; and the dizziness noted during oxygen toxicity and sensory deprivation. Here, we offer a modification of this classification, which separates diving otologic injuries into those with usually transient otologic dysfunction and those with permanent otologic inner ear injury.

Alternobaric vertigo is a short spell of vertigo, which usually occurs during ascent, and is thought to arise because of asymmetric pressure in the middle ears during equalization. The lifetime prevalence in divers has been reported to be close to 30% (14). Other causes of vertigo in diving are inner ear barotrauma, DCI, caloric stimulation, nitrogen narcosis or, in deep diving, the high-pressure nervous syndrome (11). Strong vertigo may lead to panic and irrational behaviour, which may be dangerous while under water. The diver may run out of air if forced to delay ascent in order to eliminate vertigo. Vomiting underwater may cause suffocation if wearing a helmet with an oronasal inner mask. In spite of this, no dangerous situations caused by alternobaric vertigo were reported by the divers in this study. This is in agreement with a study by Klingmann et al. (14).

Table 1. Causes of vertigo in divers

Decompression sickness
Hypoxia
Hypercarbia
Nitrogen narcosis
Seasickness
Alcoholic hangovers
Sensory deprivation
Hyperventilation
Impure breathing gas
Unequal caloric stimulation
Difficulties with middle ear pressure equilibration

Alternobaric vertigo is a specific type of middle ear barotrauma that occurs if the reduction of middle ear pressure is not equivalent in the bilateral middle ear spaces. If the pressure differential exceeds a threshold of 45 mmHg, asymmetric stimulation of the labyrinths will occur, resulting in what is called alternobaric vertigo (16). Symptoms include nausea, vomiting, disorientation, and generalized malaise. The sensation of vertigo may persist for 1 to 2 hours after ascent but gradually disappears without therapy. Divers may be particularly susceptible to alternobaric vertigo if they have had previous injury or infection of either labyrinth. In susceptible divers, use of moderate doses of antihistamines or decongestants prior to diving may prevent symptoms, although these medications are not recommended for routine use before diving as they may potentiate reverse squeeze. Although symptoms are usually mild, they may occur underwater, which is a serious problem that may lead to aspiration and drowning (18). The disorder must be differentiated from inner ear decompression sickness, which is described later and is usually associated with deeper, prolonged diving.

Transient symptoms suggestive of vestibular system dysfunction (vertigo, dizziness, and nausea) have been reported in association with high-pressure nervous syndrome (2,4). Later studies (1,3,9,10) have indicated that these symptoms are not accompanied by demonstrable electronystagmographic nystagmus but are associated with decrements in postural equilibrium and bilaterally equal increases in

the vestibulo-ocular reflex. Thus, such symptoms during high-pressure nervous syndrome are thought to be related to dysfunction in more centrally located structures and not to unilateral end-organ or primary vestibular neuron dysfunction.

Motion sickness is commonly experienced during sea, air, and car travel and is characterized by nausea, vomiting, cold sweats, yawning and hyperventilation, and pallor. The current accepted theory of neural input mismatch involves differences between real sensory input from the proprioceptive, vestibular, and visual systems compared with expected sensory input patterns to the central nervous system, which were programmed while the patient learned to crawl and ambulate during infancy (21).

Divers commonly experience motion sickness on the boat or in the wave motion zone during decompression. Most symptoms resolve upon entering the water and with descent. Habituation from continuous exposure can develop in 2 to 3 days. Nervousness, female gender, young age, and dehydration from overindulgence in alcohol have all been cited as contributors to motion sickness (6). Positioning oneself amidships while concentrating on the horizon has been suggested to help prevent or reduce the severity of symptoms.

As a diver descends and ambient pressure increases, progressively higher pressure gas is delivered to the lung and more inert gas dissolves in the blood stream. The amount of inert gas that dissolves in a given tissue is proportional to the maximum depth and bottom time, as well as the perfusion and diffusion characteristics of that tissue. As a diver ascends, the additional inert gas load comes out of solution at the level of the alveolus and is exhaled. If the rate of ascent exceeds the rate of alveolar gas exchange, inert gas will dissolute inside the diver, forming bubbles within the circulation and in tissues. The severity and nature of the decompression sickness injury vary from mild systemic, musculoskeletal and cutaneous manifestations to severe, life-threatening central nervous and cardiorespiratory symptoms.

A recent study showed that vestibular symptoms were more common in retired offshore divers than in age-matched controls (12). The symptoms were related to a previous history of DCI, which is not surprising, given the fact that vestibular involvement has been reported to be relatively common in DCI (20). Some residual symptoms are common after vestibular lesions. In the absence of manifest DCI, one might speculate whether repeated minor traumas to the inner ear may accumulate and give rise to permanent symptoms (13). This would be important since vestibular symptoms often have a significant negative impact on quality of life (19,25).

Long-term vestibular symptoms have been demonstrated in retired offshore divers (12). In contrast to this, no evidence of long-term vestibular effects was found in this study. Further follow-up of the younger divers is desirable.

Klingmann et al. (16) found that the lifetime incidence of DCI increased with decreasing diving experience. Possibly, a certain diving frequency is required in order to achieve and retain the skills necessary to avoid acute injuries.

A study found that military divers, even without a history of DCI, had more white-matter lesions on brain magnetic resonance imaging than nondiving controls (8). The same article also reviewed the evidence from previous conflicting studies. Without a dose–response relationship, it is difficult to determine whether injuries are due to the diving or to other factors that separate divers from non-divers. Such a relationship has been found between diving exposure and hyperoxic lung damage (23), but the inner ear appears to be less sensitive than the lung to hyperoxia (18).

CONCLUSION

In conclusion, transient dizziness and vertigo is common during and shortly after diving. Although these symptoms appear to be mostly benign, they may be difficult to distinguish from inner ear barotrauma and DCI. Diving carries a definite risk of acute injuries, and a high degree of vigilance is necessary to prevent these from happening. However, there was no indication in this study that frequent diving per se, in the absence of acute injuries, leads to permanent vestibular dysfunction.

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