Effects of diving on hearing and balance

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Abbreviations

DCS **Decompression sickness:** a disorder caused by gas bubble formation in the body, which occurs due to a rapid decrease in ambient pressure. Synonyms: diver's disease, caisson disease, 'the bends'.

HPNS **High-pressure neurological syndrome:** the effects of high ambient pressure (usually > 150 msw) on the nervous system. Common symptoms are tremors, nausea, dizziness and decreased psychomotor performance. Synonym: helium tremors.

Pa **Pascal:** the SI unit of pressure, which is equal to the force of one Newton acting perpendicularly on a surface measuring one square meter

Atm **Atmosphere:** a unit of pressure equal to 101 325 Pa

ATA **Atmospheres absolute:** a unit of pressure equal to 101 325 Pa. The ATA is absolute because it is defined in relation to the pressure of a vacuum. Other units may be gauge pressure (relative to the surrounding atmosphere) or absolute depending on the situation.

Bar **Bar:** a unit of pressure equal to 100 000 Pa

Msw **Meters of seawater:** a unit of pressure equal to 10 000 Pa relative to the atmospheric pressure at sea level.

Introduction

Man has always wanted to access the seabed, whether to recover persons or lost objects, to reap from its rich natural resources or sometimes—for sheer curiosity—to discover its hidden secrets. There are today millions of recreational divers worldwide, and trained divers are used for a wide range of jobs in the media, police, fisheries, military, oil industry, construction industry and more.

All this activity has important medical implications. Divers are in regular contact with the health services, usually to assess their fitness to dive, sometimes due to acute injuries and occasionally due to concerns about long-term health effects of diving. It is therefore necessary for physicians to understand how diving affects the human body.

Ear injury is the most common medical problem in diving. Historically, divers have also acquired a reputation of being hard of hearing:

> *Divers have always been deaf, so the story goes.*
> *R.C. Bornmann [52]*

However, it has proven difficult to determine whether there is truth to that story. Some studies have found that professional divers have as good or even better hearing than non-divers.

Otto Inge Molvær made a significant contribution to this field by showing that hearing in professional divers deteriorated more rapidly than in non-divers. The fact that the divers routinely used extremely noisy tools when working under water was found to be an important reason for the hearing loss, but some of them had also suffered acute inner ear injuries. It was then, and still remains, unclear whether diving itself causes hearing loss.

Molvær also focused attention on another important aspect of inner ear function, the sense of equilibrium. Acute ear injuries may cause vertigo, nausea and unsteadiness,

and people with inner ear disorders tend to find these symptoms more debilitating than hearing loss. Already in the 1890s there were detailed reports of caisson workers who were vomiting and ‘reeling like drunkards’ [31] after ear injuries. The fact that these symptoms may be caused by acute barotrauma and decompression sickness is now well known, yet there exist but a few studies on long-term vestibular symptoms in divers.

Stein Helge Glad Nordahl measured the effects of absolute pressure on postural sway systematically under hypo- and hyperbaric conditions and found that pressures both higher and lower than the pressure at sea level lead to increased postural instability. In deep diving, balance problems seemed to occur particularly at pressures greater than 200 msw.

The present thesis extends this work with a simulated saturation dive to 240 msw and two epidemiological studies on the long-term consequences of diving on both hearing and balance.

Abstract

Diving has profound effects on the human body including the inner ear, which contains the organs of hearing and balance. Ear injury is the most common medical problem in diving. The aim of this work was to explore the short and long-term effects of diving on hearing and balance through a simulated deep saturation dive and two epidemiological studies on commercial divers.

The first study explored short-term effects of absolute pressure on the body. The high-pressure neurological syndrome (HPNS) occurs at pressures exceeding 150 msw. The symptoms include tremors, decreased psychomotor performance, dizziness, nausea and drowsiness, and there is a tendency for adaptation, since many of the symptoms disappear quickly when pressure is held constant. In this study we measured postural stability in a simulated saturation dive in helium-oxygen to 240 msw, which lasted for 19 days. Eight subjects participated. Postural balance was measured 152 times during the dive by means of a force platform (static posturography). In spite of a long compression phase (>20 h) including three stops for adaptation, postural instability was observed throughout the bottom phase. There was no significant adaptation, but balance normalized during the decompression. Postural instability therefore seems to be related not only to compression rate, but also to absolute pressure. This could indicate that posturography is more sensitive than other methods in detecting HPNS.

The second study included 230 offshore divers who had been working on the Norwegian continental shelf before 1990. Most of them had retired from diving. The main finding was that these divers had more vestibular symptoms, such as dizziness, vertigo and disequilibrium than age-matched controls, and that they also had more postural instability as measured by static posturography. The finding is important, since these symptoms often lead to decreased quality of life. The symptoms were strongly associated with a previous history of decompression sickness, which is one of the major causes of morbidity in professional divers.

The third study included 67 young subjects attending a basic course for professional divers. They were examined at the course, after three and six years. Transient dizziness or vertigo was a common experience while diving, and the possible causes are discussed in this thesis. We found no long-term effects of frequent diving per se on balance or subjective disequilibrium. There were no cases of inner ear barotrauma or inner ear decompression sickness during follow-up. However, there was a progressive deterioration of hearing thresholds at 4 kHz, a frequency commonly affected by noise. There was also an increase in the prevalence of subjective hearing difficulties. Both were associated with occupational noise exposure, but not significantly with the amount of diving. We therefore concluded that noise was the most important cause of hearing loss in this diver group.

Summing up the results from the three studies, long-term effects were found on both hearing and balance, which were associated with noise and decompression sickness respectively. No long-term effects were found in association with frequent diving per se, however transient postural instability appears to be a feature of deep diving, and was found to last through the bottom phase even after a slow compression.

List of publications

I. Goplen FK, Aasen T, Nordahl SH. Postural control in a simulated saturation dive to 240 msw. Undersea Hyperb Med 2007; 34: 123-30.

II. Goplen FK, Grønning M, Irgens A, Sundal E, Nordahl SH. Vestibular symptoms and otoneurological findings in retired offshore divers. Aviat Space Environ Med 2007; 414-9.

III. Goplen FK, Grønning M, Aasen N, Nordahl, SH. Vestibular effects of diving—a 6-year prospective study. Occup Med (Lond) 2010; 60: 43-8.

IV. Goplen FK, Aasen T, Grønning M, Molvær OI, Nordahl SH. Hearing loss in working divers. Accepted for publication in European Archives of Oto-rhino-laryngology.

Contents

1. Background

This section gives a brief overview of otological aspects of diving that have relevance for the present study with focus on known effects on hearing and balance. A more general review of the physics of diving medicine, as well as of barotrauma and decompression sickness, may be found in general textbooks of diving medicine [10, 13, 19].

1.1 Inner ear barotrauma

Ear 'squeeze' or barotrauma is the most common injury in diving [74]. A study of 103 divers immediately following a single dive found that 68% had otoscopic signs of middle ear barotrauma ranging from hemorrhage around the malleus (24%) to complete hemotympanum (10%) [39]. However, it is not known to which degree these injuries may cause permanent damage to the inner ear. A study of 709 experienced US and Australian scuba divers found that the self-reported lifetime prevalence of inner ear barotrauma was 1% [74]. However, the true incidence could be higher since minor injuries might not be reported. Several cohort studies have found progressive hearing loss in working divers [24, 50, 70, 80]. A possible explanation is that repeated minor barotraumas may cause progressive damage to the inner ear [80].

The exact pathogenesis of inner ear barotrauma is usually unknown. The literature mentions several possible mechanisms including perilymphatic fistula due to rupture of the round or oval window membranes, pneumolabyrinth, rupture of Reissner's membrane and intralabyrinthine hemorrhage [47, 60]. *Round window fistulas* are sometimes found during explorative tympanotomy in divers with acute sensorineural hearing loss [60]. During compression a pressure difference may develop between the inner and middle ear. The diver feels pain in the ear, and the membrane of the round window bulges into the tympanic cavity. In this situation many divers will resort to the Valsalva maneuver, which increases the intracranial pressure and may cause the

round window membrane to rupture. There are also reports of *oval window fistulas* [47, 67] and even stapes footplate fractures [79], although these structures are considerably stronger than the round window membrane [40]. *Pneumolabyrinth* may be detected on high-resolution CT images of the temporal bone, and has been reported after direct traumas to the stapes [77]. In diving, gas expansion during ascent could force gas from the middle ear through an existing fistula and into the labyrinth. Since a fistula *per se* does not produce hearing loss [38], pneumolabyrinth could partially explain why the symptoms often start during or after ascent. After a round window rupture, pneumolabyrinth would start in the basal turn of the scala tympani producing high-frequency hearing loss, but not necessarily vertigo. *Rupture of Reissner's membrane* may explain hearing loss in the absence of a perilymph fistula [38]. This membrane consists of only two layers of flattened epithelium separated by a basal lamina, and ruptures at quite low pressures [40]. Endolymphatic hydrops, distension, collapse or rupture of Reissner's membrane has been demonstrated in guinea pigs after injection of fluid into the subarachnoid space or suction of perilymph through the round window membrane [54]. *Intralabyrinthine hemorrhage* has been suggested as a mechanism for hearing loss in divers with ear barotrauma. Such lesions are in some cases detectable as T1 or FLAIR-hyperintensities on MRI [64].

The most characteristic clinical feature of inner ear barotrauma is sudden sensorineural hearing loss, either alone or accompanied by tinnitus or vestibular symptoms. Vestibular symptoms are occasionally the only sign of barotrauma (table 1.1).

Table 1.1 Symptoms in 76 cases of inner ear barotrauma [47].

	n	%
Cochlear and vestibular	39	51
Cochlear only	30	39
Vestibular only	7	9
Sum	76	100

The symptoms of inner ear barotrauma are often noticed after the dive (table 1.2), which in some cases makes it difficult to distinguish barotrauma from decompression sickness. Symptoms occurring during ascent may be due to pneumolabyrinth. The observation that symptoms often arise directly after surfacing may indicate that it is more difficult to notice hearing loss when the head is immersed in water.

Table 1.2 Onset of cochlear and vestibular symptoms in 76 divers with inner ear barotrauma [47].

	Cochlear	%	Vestibular	%
Onset				
Descent	2	4	3	8
Bottom	6	12	7	19
Ascent	5	10	3	8
Surface	37	74	24	65
Subtotal	50	100	37	100
Missing	20		15	
Total[a]	70		52	

[a] There were 122 reported symptoms

The diagnosis of inner ear barotrauma usually requires sensorineural hearing loss documented by pure tone audiometry. Loss of otoacoustic emissions is a less specific sign, since it also occurs in middle ear disorders. In cases of vestibular involvement, objective signs include nystagmus toward the healthy side and canal paresis, lateropulsion and pathological head impulse test toward the injured side. Such signs are often absent and occur more commonly in cases of inner ear decompression sickness [37]. When present, they tend to disappear over time except canal paresis, which is sometimes permanent [37, 68].

Otoscopic signs of middle ear barotrauma are sometimes absent, since divers may seek medical advice days, weeks or months after the injury [47]. Acute signs include congestion, retraction, hematoma and perforation of the eardrum as well as blood,

exudate and gas bubbles in the tympanic cavity. These findings may be graded according to the scale introduced by Teed (table 1.3).

Table 1.3 The Teed scale of middle ear barotrauma [75]

Grade 0	Normal
Grade 1	Retraction with redness in Shrapnell's membrane and along the manubrium
Grade 2	Retraction with redness of the entire myringa
Grade 3	Same as grade 2 plus evidence of [serous] fluid in the tympanum
Grade 4	Hemotympanum, perforation of the myringa, or both

During surgery, perilymph is sometimes observed oozing from the round or oval window. In rare cases there may be stapes dislocation or fracture. Some authors advocate early exploration and closure of perilymphatic fistulas in order to improve the prognosis [33, 62]. However, others argue that spontaneous recovery is the rule, and therefore recommend expectancy [4]. To date there is no evidence-based solution to this dilemma. A study of experimental round window fistulas in guinea pigs did not find any difference in hearing outcome, based on electrocochleography, between those left open and those closed surgically [59]. Reports in humans consist mostly of small case series. Perilymphatic fistulas are notoriously difficult to diagnose. Symptoms and objective findings vary. When it comes to observing a perilymph fistula directly, even the eye of the best surgeon has unknown sensitivity and specificity, since there is no gold standard for comparison. The effect of surgery is difficult to determine in individual cases since both vestibular and cochlear symptoms tend to improve over time regardless of treatment.

Inner ear barotrauma often results in a permanent sensorineural deficit in the form of hearing loss or caloric hyporeactivity [37, 68]. Central vestibular compensation ensures a rapid recovery from most of the vestibular symptoms such as vertigo, lateropulsion and nausea. However, a more subtle feeling of disequilibrium, particularly accompanying rapid head movements, may persist.

1.2 Alternobaric vertigo

Alternobaric vertigo is a short spell of vertigo caused by increased middle ear pressure. It is experienced occasionally by 30–40% of professional divers [49] and 10–30% of military pilots [71] while ascending or performing the Valsalva maneuver.

The pathophysiology of alternobaric vertigo is not fully understood, but several studies indicate that it is related to increased middle ear pressure. Pressure applied unilaterally to the external auditory canal provokes vertigo and nystagmus in some patients with chronic tympanic membrane perforations. One study found transient nystagmus beating toward the pressurized ear [58]. The direction reversed after a few seconds. Repeated stimulations produced little or no effect. There was no evidence of a perilymph fistula and the authors reasoned that the reaction could be due to increased compliance of the bony labyrinth, so that pressure applied to the round and oval window simultaneously induced movement of the inner ear fluids, possibly by forcing perilymph through the cochlear aqueduct. The pressure (P_v) required to produce vertigo and nystagmus varied between individuals. In some subjects it was as low as 7 kPa, but more commonly 13–16 kPa.

In 1965 Lundgren raised attention to the phenomenon of transient vertigo in sports divers when ascending and called this alternobaric vertigo [44]. Further research by Tjernström, Igelstedt and co-workers reproduced vertigo and nystagmus in 2 out of 79 otologically healthy subjects both when decreasing the ambient pressure or increasing the middle ear pressure by applying pressure to the external auditory meatus after myringotomy [26, 76]. These studies showed unambiguously that it was the pressure on the round and oval windows, not the lateral displacement of the eardrum that caused the vertigo.

The forcing pressure (P_e) of the Eustachian tube is in otologically healthy subjects typically 3–5 kPa [26]. When the middle ear pressure exceeds this level, the tube opens passively and lets gas out of the middle ear. This prevents the middle ear pressure from rising to a level causing vertigo. However, P_e varies both between subjects and within subjects over time. It may be elevated due to mucosal swelling. If

the forcing pressure is higher than the pressure required to produce vertigo, then alternobaric vertigo may occur during ascent from a dive. In a study of 79 healthy divers, two of the divers with a slightly elevated forcing pressure (approx 6 kPa) demonstrated vertigo and nystagmus toward the stimulated ear [26].

Alternobaric vertigo therefore depends on two pressure thresholds that vary between individuals, the pressure required to produce vertigo and the forcing pressure of the Eustachian tube (P_e). A low P_v or a high P_e may both be predisposing factors. Vertigo also depends on pressure asymmetry. If the middle ear pressure is elevated equally in both ears, as may occur when both Eustachian tubes are blocked during ascent from a dive, vertigo may be absent.

The symptoms of alternobaric vertigo are vertigo, dizziness, nausea and occasionally vomiting while ascending from a dive, in an airplane or when performing the Valsalva maneuver. The symptom severity varies. Whether the symptoms represent a diving hazard is disputed. A review of more than 530 dives with symptoms of alternobaric vertigo did not disclose any dangerous or life-threatening situations [35]. However, in a few divers, the symptoms may be strong enough to pose a risk, e.g. when a diver vomits while wearing a helmet with oronasal inner mask [49].

The diagnosis of alternobaric vertigo is established retrospectively based on a typical history of transient vertigo of sudden onset related to a situation with increased middle ear pressure. In typical cases there are no objective signs of inner ear pathology.

There is no treatment for the condition, but the symptoms are in most cases preventable. When vertigo occurs, the diver may have difficulty reaching the surface within planned diving time. It may be necessary to descend until symptoms disappear. Subsequently, slow ascent while clearing the ears carefully (swallowing, Toynbee maneuver) is recommended. Diving with mucosal swelling in the ears and nose (e.g. with common cold or allergy) should be avoided. Nasal decongestants are usually not recommended since the effect is short-lasting and the symptoms usually occur during ascent.

1.3 Inner ear decompression sickness

Decompression sickness (DCS) sometimes causes injury to the inner ear [20, 37, 53]. The incidence is unknown, but several published case series [32, 37, 53] indicate that these injuries are encountered regularly in centers that treat divers with DCS. The symptoms and findings are quite similar to those of inner ear barotrauma, and the differential diagnosis may be difficult. Historically, there has therefore been some doubt whether isolated inner ear DCS is a real disease entity or represent misdiagnosed barotraumas [18]. However, animal studies support the theory that DCS can produce discrete inner ear injuries [42, 43, 78]. In a study of experimental DCS in squirrel monkeys a diving profile produced isolated vestibular 'hits' in 35% of attempts [41]. Bilateral myringotomy was performed before the dives to avoid ear barotrauma. The diagnosis of inner ear DCS was confirmed histologically.

Given these findings, the inner ear seems to demonstrate a particular susceptibility to DCS. Nachum et al reported that inner ear injuries constituted 26% of all their cases of serious DCS in scuba divers [53]. A review of 2500 US Navy diving accidents found vestibular involvement in 10–20% of DCS cases [32]. In humans, such injuries are associated with the presence of a patent foramen ovale [14, 34]. Shunting of gas emboli from the venous to the arterial circulation increases the risk of disturbed cerebral circulation, but this does not explain the apparent susceptibility of the inner ear. Local factors must therefore be of importance, such as inert gas supersaturation and low tissue compliance [16].

The pathology of inner ear DCS has been described in squirrel monkeys and includes hemorrhage, amorphous endolymph precipitate, cupular deposits and even semicircular canal fractures [21, 42]. The endolymph precipitate and cupular deposits may explain why some of the monkeys demonstrated positional nystagmus. The clinical significance of microfractures in the temporal bone is disputed [22].

The symptoms of inner ear DCS include dizziness, vertigo, nausea, vomiting and falling. The onset is acute during ascent or shortly after the dive, often with a symptom-free interval.

The clinical signs of vestibular DCS are lateropulsion to the injured side, nystagmus toward the healthy side, canal paresis and a pathological head impulse test toward the injured side. When present, a cochlear lesion is characterized by sensorineural hearing loss.

In clinical practice inner ear decompression sickness has many features in common with inner ear barotrauma, and the differential diagnosis is difficult (table 1.4). In typical cases of inner ear DCS the symptom onset is during ascent or after surfacing, and the diving depth or time exceeds the limits requiring decompression stops according to standard diving tables. In monosymptomatic cases, vestibular symptoms are more common than cochlear [37, 68]. Characteristically, pressure equalization problems during ascent or otoscopic signs of barotrauma are absent. However, none of these features are in themselves diagnostic.

Table 1.4 Clinical features[a] that may differentiate between inner ear decompression sickness and inner ear barotrauma.

Inner ear decompression sickness	Inner ear barotrauma
Symptom-free interval after the dive	Onset of symptoms during the dive
Dive depth or time exceeding table limits	Problems clearing the ears during compression
Vestibular lesion without hearing loss (spontaneous nystagmus, pathological head impulse test, canal paresis)	Otoscopic findings indicating ear barotrauma
	Isolated sensorineural hearing loss (tuning fork tests or audiometry) in the absence of vestibular symptoms or signs

[a] None of which should be considered ‘pathognomonic’ or ‘diagnostic’

Suspected inner ear decompression sickness is treated with oxygen and recompression. Subsequent treatment may include hearing aids and vestibular rehabilitation.

As in inner ear barotraumas, residual pathology is common, including sensorineural hearing loss and canal paresis, the latter being accompanied in some cases by light sensations of disequilibrium with head movement [37]. Farmer reported good results from recompression within 42 minutes with no residual inner ear dysfunction [20]. However, peripheral vestibular deficits have a good prognosis due to central compensation, and other studies have indicated that these deficits tend to persist in spite of early recompression therapy [41].

1.4 Nitrogen narcosis

In 1834 Junod reported behavioral changes similar to mild alcohol intoxication in patients breathing compressed air [30]. Later authors observed drowsiness, hallucinations and impaired judgment in deep dives [7]. In 1935 Behnke argued correctly that this was a narcotic effect of nitrogen [5]. The symptoms are reversible, but may be dangerous while the diver is in the water. Today nitrogen narcosis is a well-known phenomenon, and divers are advised against diving beyond 30-50 msw when using air as breathing gas.

Nitrogen narcosis may present itself with symptoms that resemble vestibular disorders. Adolfson et al noted that divers ‘repeatedly reported feelings of dizziness and vertigo during exposure to increased atmospheric pressure while breathing certain gases or gas mixtures’ [1]. The same author measured standing steadiness [3] and nystagmus induced by passive constant velocity head rotations [2] in divers breathing air at 0 and 90 msw. At 90 msw severe postural instability was observed, but nystagmus frequency and amplitude was only slightly decreased. The authors argued that the decrease was insufficient to explain the severity of the symptoms, and that these were more likely of central origin, i.e. due to nitrogen narcosis.

1.5 The high-pressure neurological syndrome

Replacing nitrogen in the breathing gas with other inert gases such as helium and hydrogen makes it possible to avoid nitrogen narcosis. However, at high pressures other problems arise. The high-pressure neurological syndrome (HPNS) starts at pressures exceeding 150 msw with tremors, dizziness, nausea and impaired psychomotor performance. The severity of the symptoms varies between individuals and can be reduced considerably by using very slow compressions with frequent stops. In this way human divers have tolerated pressures up to 701 msw. Nevertheless, HPNS seriously limits the ability of man to tolerate deep diving. At greater pressures there may be drowsiness, memory lapses (‘micro sleep’), momentary loss of consciousness and hallucinations. In mice convulsions and death occur at pressures from 900 to 1300 msw [65].

The effect of high pressure has been likened to that of cooling [72] in that chemical processes are slowed down resulting in reduced excitatory synaptic activity in the brain. This reduction is partially compensated for by an increased excitability on the dendritic side. However, the mechanisms behind HPNS are still not fully understood.

Other symptoms associated with HPNS are vertigo, nausea, vomiting and postural instability. Because these symptoms are also characteristic of peripheral vestibular disorders, they raise the possibility that the vestibular system is affected by absolute pressure. Bennett noted in 1965 that the loss of psychomotor efficiency in divers breathing helium-oxygen at 180–240 msw was accompanied by ‘dizziness, nausea and tremor of the hands, arms and even the whole body’ [6]. Braithwaite et al measured postural sway as well as spontaneous, caloric and optokinetic nystagmus in divers breathing helium-oxygen at 485 msw [12]. There was a marked increase in postural sway, but no vestibular symptoms were reported. There was no spontaneous nystagmus. Nystagmus induced by optokinetic and caloric stimulation was slightly diminished in amplitude, but within normal limits. Molvær measured the caloric response in eight divers during the bottom phases of two dives to maximum pressures of 300 and 504 msw [47]. At 300 msw, the caloric response was reduced in all divers,

however this finding was only reproduced in two out of six divers in the deeper dive. Renon found decreased caloric response in six out of eight divers breathing trimix at 450 msw [63]. However, Gauthier found increased response to passive sinusoidal rotations in two divers at 610 msw [23]. Thus, the majority of the studies indicate a small and variable depressive effect of high ambient pressure on the gain of the vestibulocular reflex (VOR). Drowsiness is one of the symptoms in HPNS. The gain and velocity storage of the VOR depends on alertness [46]. A possible depressive effect of HPNS on the VOR could therefore be related to drowsiness.

Nordahl et al found increased postural sway in divers breathing helium-oxygen at 100 and 450 msw [55, 56]. In the deeper dive, 200 msw seemed to be a critical level in the development of postural sway. One of the aims of the present study was therefore to explore this level more closely.

1.6 Oxygen toxicity

Oxygen at high partial pressures is toxic to all living organisms [8]. In humans acute oxygen poisoning mainly affect the central nervous system with muscle twitching, particularly in the lips, clumsiness and convulsions [15], while prolonged exposure causes damage to the lungs and eyes [61]. The acute effects mainly occur when the partial pressure of oxygen exceeds 2 ATA or 200 kPa.

Vertigo is a known symptom of acute oxygen poisoning both during exposure and after withdrawal (‘oxygen-off effect’) [18]. In addition there is often nausea and sometimes vomiting. Donald reported the acute effects of oxygen poisoning in 36 healthy subjects. Five subjects had vertigo and five experienced nausea or vomiting. A study of cochleovestibular function before, during and after exposure to oxygen at 3 ATA did not find any changes in audiometry thresholds. Caloric nystagmus was unchanged after exposure. The limited evidence, therefore, does not indicate any short-term effects of hyperbaric oxygen on peripheral cochleovestibular function. Vertigo, nausea and vomiting in acute oxygen poisoning are therefore more likely

due to an effect on the central nervous system. There are to date no studies on the role of oxygen in long-term cochleovestibular function in divers.

1.7 Long-term hearing loss in divers

The high incidence of ear injuries in divers makes it natural to ask whether diving causes long-term hearing loss. This question is more difficult to answer than one would expect, and different studies have given equivocal results. However, some facts are generally agreed upon. First, the relationship between occupational noise and hearing loss is well known, and professional divers are exposed to high noise levels from a number of sources including gas valves, propellers, explosions and underwater tools [48, 51, 69]. Second, short and long-term hearing loss after acute diving-related ear injuries is well known and documented in many studies [37, 47, 68]. Third, there are now several independent cohort studies showing progressive hearing loss in working divers [24, 50, 70, 80]. Based on this, one would suspect that divers are at increased risk of hearing loss. However, several studies have found that divers' hearing is comparable to or even better than age-matched controls [11, 25, 36, 52, 73]. There may be several reasons for this.

Divers start out with better-than-average hearing

Molvær et al suggested that professional divers start their career with better-than-average hearing because persons with ear disorders are less likely to become divers [52]. Later in life the divers' hearing thresholds increase so that they at some point pass the population mean. This could in part explain why some cross-sectional studies have failed to find differences between divers and non-diving controls.

Noise exposure

Underwater tools may generate extreme noise levels [69]. Based on health and safety recommendations on land, there is little doubt that these noise levels are capable of producing long-term hearing loss, and studies of divers have found temporary

threshold shifts after the use of high-pressure water jets and rock drills under water [51]. It is therefore a possibility that most of the hearing loss in divers is due to noise exposure. This could explain the fact that no studies of recreational divers have documented hearing loss compared to controls [25, 36, 73].

Acute injuries

Inner ear barotrauma and inner ear decompression sickness may lead to permanent hearing loss. A study of recreational divers found that approximately 1% had experienced inner ear barotrauma [74]. However, if this incidence is correct, such injuries should have little influence on hearing in the general diver population. Even a deaf ear in 100 divers (200 ears) would have little effect on mean hearing threshold in the diver population and no effect on the median.

The incidence of inner ear injuries may be much higher in some subpopulations of divers. Edmonds found a high prevalence of moderate to severe sensorineural hearing loss in Australian abalone divers [17]. However, most studies of occupational divers have shown more moderate, if any, hearing loss [11, 52, 70, 80].

Repeated minor injuries

A simple ‘ear squeeze’ or middle ear barotrauma may cause conductive hearing loss. This condition usually resolves spontaneously. However, it occurs so frequently in diving that the question arises whether repeated minor injuries in the long run might lead to permanent hearing loss. In theory, multiple injuries could lead to fibrosis, decreased mobility of the ossicular chain and conductive hearing loss. Repeated minor unnoticed injuries to the inner ear could also lead to long-term sensorineural hearing loss. One of the purposes of this study was do examine a group of professional divers prospectively in order to discover signs of progressive hearing loss or vestibular disorders.

1.8 Long-term vestibular disorders in divers

The high incidence of ear injuries in divers could have implications for vestibular function. Chronic vestibular symptoms include vertigo, dizziness or a feeling of disequilibrium, particularly with head movements, postural instability and oscillopsia. Such symptoms may have a considerable negative impact on quality of life. Yet there are but a few available studies on long-term vestibular effects of diving. Sharoni et al compared 13 asymptomatic professional divers to 12 non-divers [66]. The only difference found was a slightly shorter vestibulo-ocular reflex (VOR) phase lead in the divers. The authors suggested that this could be an adaptation in the divers to the underwater environment augmenting the low-frequency response of the VOR. Macdiarmid et al measured postural sway in a large study of occupational divers and found no difference between the divers and controls [45]. It is therefore to date no evidence that diving has any long-term adverse effects on vestibular function. A purpose of this study was to examine this question more closely both in a cross-sectional study of retired divers and in a prospective study of younger divers.

2. Aims

2.1 General

The overall aim of this thesis was to gain in-depth knowledge about the short and long-term effects of diving on hearing and balance.

2.2 Paper I

The aim of paper I was to examine the short-term effects of high pressure on balance. A previous study by Nordahl et al had found a sharp increase in postural sway at pressures exceeding 200 msw. The saturation dive to 240 msw represented an opportunity to measure this change in more detail under conditions that offered maximum control over both exposure and measurements. Variations in postural sway during the different phases of the dive were expected to throw light on when and why divers experience disequilibrium in deep diving.

2.3 Paper II

The aim of paper II was to examine the long-term effects of diving on balance. The divers who worked on the Norwegian oil installations in the North Sea before 1990 had for the most part retired from diving. These divers were of interest to us for several reasons. First, they were relatively numerous, since before 1990 divers were required to perform many of the tasks that are now accomplished by remotely operated vehicles (ROV). Second, their exposure included deep, surface oriented diving (‘bounce diving’) and a high lifetime prevalence of dysbaric conditions, particularly decompression sickness. We therefore considered it very likely that they could provide us with valuable insight into the causes and mechanisms of disturbances of hearing and balance in professional divers. We thought that this

insight would be more difficult to obtain from a study of modern offshore divers because they are fewer in numbers and mainly perform saturation dives, which carry a lower risk of barotraumas and decompression sickness.

2.4 Paper III

To complement the study behind paper II a prospective epidemiological study was desirable. A longitudinal design would give the study higher statistical power and allow more reliable collection of exposure data. By following a group of newly trained professional divers for the first six years of their career, we hoped to gain more knowledge about the causes of vertigo, dizziness and postural imbalance in divers.

2.5 Paper IV

The intention in paper IV was to use the same group of divers as in paper III and measure changes in hearing acuity during the first six years of their career. By the prospective design and quantification of exposure, we wanted to shed light on the causes of hearing loss in divers.

3. Methods

3.1 Ethics

The research protocols were approved in advance by the Regional Committee for Medical Research Ethics, which enforces the Helsinki Declaration on medical research involving humans. Participation in the studies was based on written informed consent.

3.2 Design

The first study (paper I) was an experimental study, the second (paper II) was a cross-sectional questionnaire survey with follow-up medical examination in a subset of respondents, and the third (paper III and IV) was a prospective cohort study with six-year follow-up.

3.3 Target groups

The target groups were different sub-populations of professional divers. Specifically we wanted certified saturation divers for paper I, experienced offshore divers (working on the Norwegian continental shelf before 1990) for paper II and newly trained professional divers for paper III and IV.

3.4 Subjects

The first study (paper I) included eight men aged 41 (range 29–48) years. Seven of them were certified saturation divers and one was a medically qualified scientist with experience in SCUBA and saturation diving.

The second study (paper II) included 230 men aged 52 ± 6.7 yr (mean ± SD; range 35–69 yr) in the questionnaire study and a subset of 96 men in the follow-up medical examination. A control group of age-matched men (non-divers) were drawn randomly from the population of Bergen and neighboring municipalities. Of those contacted, 166 (26%) agreed to participate. A subgroup of 42 randomly selected men went on for otolaryngological examination.

The third study (paper III and IV) included 67 men aged 28 ± 6.5 years (mean ± SD) who attended a basic course for professional divers at Statens dykkerskole (Norwegian State Diving School).

3.5 Timeline

The third study (paper III and IV) was a longitudinal study with six-year follow-up. The divers were examined while attending the diving school, and the procedure was repeated after three and six years.

3.6 Questionnaires and personal logbooks

Questionnaires were used to record essential occupational and medical history data in paper II to IV. In the longitudinal study (paper III and IV) diving exposure data were obtained also from the divers' personal logbooks. The questionnaire for the offshore divers (paper II) is found in Appendix A. The questionnaire for the cohort of younger divers (paper III and IV) is in Appendix B.

3.7 Interview and clinical examination

The clinical examinations were conducted at the Department of Otolaryngology, Head and Neck Surgery at Haukeland University Hospital by three of the co-authors (Goplen, Nordahl and Molvær).

The divers were interviewed about their diving history including type of education, type and amount of diving, noise exposure, barotraumas, decompression sickness, other injuries and health problems related to diving. The medical history was also recorded with regards to ear-nose-throat disorders and general medical disorders. Particular attention was given to otological or otoneurological symptoms or disorders.

The clinical examination included a general otolaryngological examination, testing of the cranial nerves, coordination (diadochokinesis, finger-to-nose pointing and tandem gait), balance (Romberg's test and step test) and nystagmus (spontaneous or provoked by lateral gaze, headshake or the Hallpike maneuver).

3.8 Electronystagmography

All subjects underwent electronystagmography with test for spontaneous and positional nystagmus, ocular smooth pursuit and saccadic pursuit and bi-thermal caloric response. Abnormal spontaneous nystagmus was defined as nystagmus with slow phase velocity > 5 degrees per second. Unilateral caloric weakness was defined as a side difference > 25% according to Jongkees' formula [29].

3.9 Static posturography

In order to measure standing steadiness, we used a commercially available force platform (Cosmogamma®, Italy) measuring 40 x 40 cm with three strain gauge pressure transducers.

Postural sway was measured in four different test conditions. In paper II and III measurements were taken with eyes open (EO) and closed (EC). In paper I sway was also measured while the subjects were standing on a 10 cm thick foam rubber mat that was placed on top of the platform. Each measurement lasted 60 s, except in paper III where the measurements lasted 180 s.

The center of pressure (COP) under the soles of the feet was sampled by the platform

at a rate of 10 Hz. The movements of the COP reflected the corrective forces exerted on the platform by the subject in order to maintain steady posture. The length of the curve (path length) described by the COP was used as test variable for statistical evaluation.

3.10 The 240-msw dive

This dive was a simulated saturation dive with helium-oxygen as breathing gas. It lasted 19.3 days with 20 h compression phase, 6.6 days bottom phase and 11.9 days decompression phase. The saturation pressure was 240 msw. There were excursions to 250 and 254 msw. Oxygen partial pressure varied from 35 kPa to 70 kPa.

Static posturography was performed twice daily throughout the dive with the exception of three tests that were cancelled due to other activities. We obtained 4×38 measurements during the whole dive.

Caloric responses were also compared before and after the dive. For this purpose the maximum slow phase velocity after the four irrigations were added and divided by four.

3.11 Measurement of hearing

Hearing was measured at the university clinic in soundproof rooms and by trained audio technicians. Pure tone audiometry with air and bone conduction was measured in the frequencies 0.5, 1, 2, 3, 4 and 6 kHz. Middle ear pathology was assessed by otomicroscopy, impedance tympanometry and measurement of air-bone gap.

3.12 Calculation of hearing loss and threshold shift

The Statistical distribution of hearing thresholds as a function of age (ISO 7029) in otologically normal subjects [27] was used in order to calculate age-adjusted

thresholds according to the following formula: Age-adjusted threshold = Observed threshold - Expected threshold (ISO 7029). The right and left ear values were averaged. Threshold shift was calculated by subtracting the age-adjusted threshold at diving school from the age-adjusted threshold after six years.

3.13 Statistical analysis

Statistical analysis was performed using SPSS/PASW and Stata (StataCorp, Texas, USA). The type I error level was taken to be $\alpha = 0.05$. Paper I used the runs test, paired t-tests and linear regression. Paper II used chi-square tests, t-tests and rank sum tests (Mann-Whitney). Paper III used McNemar's test, rank correlation (Kendall), signed rank tests (Wilcoxon), rank sum tests (Mann-Whitney) and chi-square tests. Paper IV used logistic and linear regression as well as multivariate repeated measures anova, t-tests and Cochran's test.

4. Summary of results

4.1 The 240 msw dive

4.1.1 Effects on postural sway

After the compression to 240 msw one diver reported vertigo, disequilibrium and nausea, while posturography showed a marked increase in postural sway. One diver was asymptomatic and displayed only random variations in postural sway (runs test: $p > 0.05$). The remaining four divers were asymptomatic, but showed mild to moderate increases in postural sway during the bottom phase of the dive. This was also true of two divers that were excluded from the main analysis due to unilateral caloric weakness before the dive. Mean postural sway increased during the compression to 240 msw, remained increased throughout the bottom phase, and returned to pre-dive levels during the decompression. The mean increase in path length at 240 msw was approximately 30%, except when the subjects were standing on bare platform with eyes closed. In this condition the increase was less consistent.

4.1.2 Effects on caloric response

There was no difference in caloric response before and after the dive.

4.2 The epidemiological studies

4.2.1 Differences in demographics and exposure

The two included diver groups differed in terms of age, professional experience and exposure to injuries (table 4.1).

TABLE 4.1. DIFFERENCES BETWEEN THE DIVER GROUPS.

	Retired offshore divers (paper II)	Younger divers (paper III)
Age (mean)	52	28
Years of diving experience (median)	12	6
Number of air dives (median)	700	320
Maximum diving depth in meters (median) *	150	60
Number of mixed gas bounce dives (median)	14	0
Days in saturation (median)	150	0
Decompression sickness (% prevalence) †	61	3

* The younger divers were certified for diving to 50 msw and had experienced diving to 60 msw at the diving course

† Type I cases reported by the divers (paper I) or all possible cases diagnosed retrospectively (paper II)

4.2.2 Prevalence of subjective symptoms

In the retired offshore divers, subjective dizziness (28%), spinning vertigo (14%) and unsteady gait (25%) were more common than in the controls. There was no difference in susceptibility to motion sickness. Apart from motion sickness, the subjective symptoms were strongly associated with a history of type I decompression sickness and less strongly with the amount of diving.

In the younger divers, there was no change in the prevalence of subjective vestibular symptoms during follow-up. However, transient vestibular symptoms during (63%) or directly after (15%) a dive were common. Alternobaric vertigo, defined as dizziness or vertigo related to pressure equalization during ascent, was the most common type of dizziness while diving (35%).

4.2.3 Prevalence of ENG-abnormalities

Abnormal positional nystagmus was present in 7% of the retired offshore divers and 5% of the controls. Canal paresis was present in 5% of the divers and in none of the

controls. Pathological ocular smooth pursuit was present in 8% of divers and 12% of controls.

In the younger divers, there were no changes in the prevalence of abnormal ENG-findings during follow-up

4.2.4 Static posturography

In the retired offshore divers, 32% had abnormal postural sway defined as path length greater than that in 95% of the control population. Abnormal postural sway was associated with reported unsteady gait, but not with subjective dizziness or spinning vertigo. Nor was postural sway associated with high diving exposure or a history of DCS.

In the younger divers, there was a small increase in postural sway during the six-year follow-up, however this was not associated with the amount of diving.

4.2.5 Clinical vestibular disorders

In the retired offshore divers a clinical diagnosis of otogenic vertigo was found in eight cases (8%) of those examined. Five cases were benign paroxysmal positional vertigo, one case was vestibular neuritis and two cases unilateral vestibulopathy of unknown etiology. In the controls one subject (2%) was diagnosed with otogenic vertigo (benign paroxysmal positional vertigo).

In the younger divers there were no diagnosed cases of vestibular disorder.

4.2.6 Prevalence of middle ear barotrauma

Among the younger divers, the lifetime prevalence of middle ear barotrauma was 36%.

4.2.7 Prevalence of inner ear barotrauma and inner ear decompression sickness

Among the younger divers, there were no diagnosed cases of inner ear barotrauma or inner ear decompression sickness during follow-up.

4.2.8 Hearing loss

In the younger divers, a permanent threshold shift at 4 kHz occurred during follow-up. There was also an increase in the prevalence of subjective difficulties understanding speech in background noise. Both the threshold shift and the subjective hearing problems were associated with noise exposure, but not with the amount of diving.

5. Discussion

5.1 The short-term effect of pressure on balance

This study confirms previous reports that increased pressure is associated with increased postural sway [12, 55, 56]. The compression was slow (>20 h) with three stops, and there was little evidence of adaptation during the bottom phase. This indicates that the measured effect is related to absolute pressure rather than to compression. The oxygen pressure was kept well below the levels considered to cause CNS toxicity [9]. There were great inter-individual differences ranging from severe vertigo, disequilibrium and nausea in one subject to no symptoms and only random variations in postural sway in another. This indicates that the finding represented a real physiological effect, not an artifact. In the remaining divers, there was measurable postural instability, but to a moderate degree.

The prospective experimental setup allowed for control over the exposure and measurement conditions. The great number of measurements makes this study the most detailed of its kind, minimizing the possibility of random variations interfering with the results.

The method of decreasing visual and proprioceptive input by eye closure and by standing on a foam rubber mat respectively has been described previously by Norré [57]. In the present dive, the most significant effects seemed to occur when standing on foam rubber with eyes closed, which is when balance is most dependent on vestibular function.

There are many causes of increased postural sway. However, when it is combined with vertigo and nausea, as has been found previously in studies of the high-pressure neurological syndrome, a vestibular origin may be suspected. Since HPNS is thought to be a general effect on the nervous system, vestibular symptoms may originate in the vestibular nuclei, which occupy a large portion of the brainstem along the floor of

the fourth ventricle. Nevertheless, the exact pathophysiology of postural sway remains unknown, and posturography cannot provide any definite answers due to its lack of specificity. The results must therefore be interpreted in light of previous studies based on different methods.

Some studies—as reviewed in chapter 1.5—have found that pressure reduces the gain of the vestibulo-ocular reflex. This could be related to general drowsiness secondary to the high-pressure neurological syndrome. This syndrome has been studied extensively, and a review is beyond the scope of this thesis. A central feature is adaptation, and several studies have found that the symptoms disappear during the bottom phase. Our findings were different in that the postural sway persisted throughout the bottom phase. Therefore, adaptation seems to be less complete with regards to postural sway. This finding may imply that posturography is more sensitive to HPNS than previous methods.

5.2 Long-term effects on balance

5.2.1 The retired offshore divers

This study is the first to show that vestibular symptoms, including vertigo, dizziness and unsteadiness, are more common in a group of retired offshore divers than in controls. The finding is important since vestibular symptoms may have a significant negative effect on quality of life [28]. The symptoms were strongly associated with a history of decompression sickness, which is a well-known cause of morbidity in divers. The symptoms were associated with an objective increase in postural sway measured by static posturography.

The study captured a large proportion (61%) of the entire population of divers working on the Norwegian offshore installations before 1990. A large proportion (44%) was also referred for clinical examination.

Nevertheless, the results must be interpreted with care due to possible biases in the material. Table IV in paper II shows referral bias, which is relevant when interpreting the results from the clinical examinations. The questionnaire survey was not affected by referral bias, since it included approximately the same proportion of referred divers as the entire target population.

Another potential problem is reporting bias. A person claiming disability might also have an interest in reporting health symptoms. However, the divers who were not claiming disability, and who were not referred to us, also reported a significantly higher prevalence of dizziness, and particularly spinning vertigo, than the controls. The symptoms were associated with a previous history of DCS and with diving exposure. The divers also seemed to be specific when indicating the type of symptoms they experienced since the divers reporting vertigo/dizziness were different from those with unsteady gait or motion sickness, and only unsteady gait was associated with increased objective postural sway. This coherence in the symptom reports indicates an underlying pathology, which cannot be explained solely by reporting bias.

The cause of the symptoms cannot be determined for sure. The high prevalence of spinning vertigo, in particular, could imply that the problem at least in some of the cases was of a vestibular nature. The most common diagnosis in the referred divers was benign paroxysmal positional vertigo. This is a common disorder in the general population, and is not necessarily related to diving. The lack of a precise vestibular diagnosis in most of the cases could be due to the fact that the symptoms were of a chronic nature, and that vestibular disorders are most easily diagnosed in the acute phase, when there are objective signs like spontaneous nystagmus and lateropulsion, and when the divers themselves remember the exact circumstances under which the symptoms occurred.

5.2.2 The young inshore divers

This study is the first prospective follow-up of a group of professional divers with regards to vestibular symptoms and disorders. No long-term effects were found.

The results may seem to contradict those from the study of retired offshore divers. However, it must be emphasized that the symptoms in the latter group were associated with decompression sickness, of which there were very few cases in the former group. In the younger divers, there were no diagnosed cases of inner ear injury during follow-up and no treated cases of DCS. Therefore, the correct interpretation is that no long-term vestibular effects were found *in the absence of acute inner ear injuries and clinical DCS*. We did not find evidence that frequent diving *per se* leads to more vestibular symptoms or to more postural sway.

This study was prospective with a careful collection of clinical, biometric and exposure data. It is therefore unlikely that significant events or signs of vestibular injury escaped scrutiny. The diver group was relatively homogenous since they had received the same comprehensive professional training. They were young, newly trained and appeared to be well motivated for the study.

No non-divers were included in the follow-up. Instead the effect of diving was analyzed by linear regression using diving exposure as the independent variable. Some of the subjects were very active diving (maximum 2000 dives) while others hardly dived at all during follow-up (minimum 2 dives). In this way the exposed and non-exposed subjects were presumably more similar than if a separate group of non-divers had been selected from the general population.

Vertigo was found to be common as a transient symptom while diving. The lifetime prevalence of alternobaric vertigo was similar to that found in two previous studies [35, 49]. Based on these findings it seems reasonable to conclude that about one in three divers is susceptible to this type of vertigo.

The finding that 15% of the divers had experienced dizziness shortly after diving was somewhat surprising. Such symptoms may indicate decompression sickness, and

should lead to immediate contact with a diving physician. The reason why the divers failed to do so could be that they interpreted their own symptoms as mild or insignificant. No cases of inner ear decompression sickness or inner ear barotrauma were diagnosed during follow-up.

5.3 Long-term effects on hearing

This study showed a progressive hearing threshold shift at 4 kHz in a group of professional divers followed prospectively for six years. This shift was associated with noise exposure. So was the increase in prevalence of subjective hearing difficulties during follow-up. No association was found between the hearing loss and the amount of diving. Therefore, we cannot from this study conclude that diving per se causes hearing loss. It must be emphasized that none of the divers had inner ear barotraumas or inner ear decompression sickness during follow-up, and that such injuries are important causes of hearing loss in divers, even if they are relatively rare. It must also be remembered that such injuries may be more common in some subpopulations of divers. Edmonds found a high prevalence of severe sensorineural hearing loss in a group of Australian abalone divers [17]. The standardized training and prospective follow-up of the divers in our study could have helped to prevent such injuries.

6. Conclusion and future perspectives

Summing up the results from the three studies, long-term effects were found of occupational diving on both hearing and balance. The main associations were found between noise and hearing loss, and between decompression sickness and vestibular symptoms. Short-lasting vestibular symptoms, including dizziness and vertigo, are common during or shortly after diving. While these symptoms are mostly benign, e.g. due to alternobaric vertigo, they may also signal injury to the inner ear. An onset shortly after ascent implies that the symptoms could be caused by decompression sickness, which requires immediate hyperbaric oxygen therapy. In this study no long-term effects were found of frequent diving per se. However, deep diving was found to be associated with transient postural instability, which persisted through the bottom phase even after a slow compression.

Diving is an activity that is particularly unforgiving of ignorance. Since the days of Paul Bert (1833–1886), who was one of the first to understand the problems of decompression sickness and oxygen toxicity, there has been a tremendous increase in knowledge within the field of diving medicine [8]. There are still many important gaps in our understanding of diving related injuries, why they occur and how they should be diagnosed and treated. On a more basic level, a greater appreciation of how pressure affects inner ear function could lead to a better understanding of other disorders such as Menière's disease, perilymph fistulas and related conditions, which remain enigmas despite decades of research.

It is unlikely that man will eventually loose interest in diving, and consequently, inner ear injuries will occur. However, with more knowledge and adequate training, there is good reason to hope that working divers growing deaf or loosing their balance after ear injuries will be a decreasing problem.

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