Susceptibility of the Inner Ear Structure to Shunt-Related Decompression Sickness

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Introduction: Decompression sickness (DCS) is caused by formation and growth of bubbles from excess dissolved gas in body tissues following reduction in ambient pressure. Inner ear decompression sickness (IEDCS) is a complex disorder involving the vestibulo-cochlear apparatus whose pathophysiology remains incompletely understood. ***Methods:*** The records of 662 consecutive DCS cases treated over a 7-yr period at 2 UK hyperbaric units were examined for symptoms suggesting IEDCS (nausea, vomiting, dizziness, and hearing loss arising within 2 h of surfacing). For IEDCS cases, demographics, dive, treatment, and outcome data were extracted with particular attention to the outcome of testing for a right-to-left shunt. ***Results:*** Included were 31 men and 2 women with a mean age of 46 yr (range 31-61 yr).Of these, 16 patients had isolated IEDCS and 17 patients had associated symptoms ranging from joint pain to tingling and numbness. The depth of the dive leading to the incident ranged from 49-256 ft (15-78 m). As primary treatment, 21 patients received a U.S. Navy Treatment Table 6 (USN TT6) and 11 patients received a Comex 30. No difference in the speed of recovery or number of treatments needed was seen between the two tables. All patients were advised to have a right to left shunt (RLS) check, but only 30 complied with that, with 24 (80%) testing RLS positive. ***Conclusion:*** Our retrospective study confirms the correlation between IEDCS and the presence of a significant patent foramen ovale (PFO). In our series 48% of patients had an isolated IEDCS. IEDCS responds slowly to treatment irrespective of the initial table used. Recovery is thought to be mainly a central compensation process.

Keywords: inner ear decompression sickness, right to left shunt, patent foramen ovale, treatment tables.

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DECOMPRESSION sickness (DCS) is caused by the formation and growth of bubbles from excess dissolved gas in body tissues following a reduction in ambient pressure. It is a complex disorder that can affect various organs and whose pathophysiology remains incompletely understood (6). One manifestation of DCS, commonly referred to as "inner ear decompression sickness" (IEDCS) presents with vestibular symptoms, including nausea, vomiting, dizziness, and/or cochlear symptoms such as hearing loss, and may occur in association with other DCS manifestations or as an isolated clinical entity (12).

Over the last 30 yr recreational diving has become increasingly popular as a leisure activity. Recreational dives were initially completed with air as a breathing mixture, but over the last decade, as a result of the desire for longer and deeper diving and penetration of deep caves and wrecks, "technical diving" has emerged. Technical divers use different gas mixtures of helium, oxygen, and nitrogen that were once only used for deep saturation diving in professional diving.

Cases of isolated IEDCS have been primarily associated with decompression from deep, mixed gas diving and have been attributed to the switch from a gas mixture containing helium, nitrogen, and oxygen to one containing only nitrogen and oxygen. The pathophysiology of the symptoms (dizziness, nausea) occurring underwater after the switch have been attributed to gas counter-diffusion. Counter-diffusion can occur when different inert gases are breathed in sequence and was first described by Idicula and Lambertsen in 1975 (10). In the inner ear it is the switch from a helium-rich atmosphere to nitrogen that was described as creating a transient supersaturation state. The model described by Doolette and Mitchell implicates transient supersaturation resulting from a counter-diffusion mechanism in which helium transfer from the perilymph to the other compartments temporarily exceeds the washout of helium in the venous blood (4). However, the gas counter-diffusion theory does not explain the occurrence of IEDCS after air dives without any gas switching or helium involvement, and there have been multiple studies published in the last decade describing isolated and combined symptoms of IEDCS after air diving (3,8,9). The biophysical basis for this selective vulnerability of the inner ear to DCS has not been established (4).

In addition to the gas counter-diffusion theory for IEDCS, several authors have proposed the involvement of a right-to-left (RLS) shunt in its manifestation, with slower inert gas washout from the inner ear playing a significant role (11). Previously published case series have found a very high incidence of a RLS of 82%, 83%, and 73%, respectively, in divers with IEDCS as compared with 25–33% in the control population. (3,7,9) Again, Mitchell and Doolette have published a theoretical

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model that shows that inert gas elimination from the inner ear is much slower than that for the brain, and this together with the presence of a RLS could explain the isolated occurrence of IEDCS (11).

But still, the number of published cases of divers with IEDCS remains small. In order to further offer interesting clinical data on this disease we have performed a retrospective analysis of 33 cases of IEDCS treated and followed up at two major hyperbaric units in the UK. The analysis explores the potential correlation between parameters such as dive profile, breathing gas mix, time to first treatment, treatment table used, speed of recovery, number of treatments needed until no improvement, recovery, and risk factors, including the presence of a RLS.

METHODS

Participants

This was a retrospective notes review of all cases of decompression illness (DCI) presenting at the Diving Diseases Research Centre (Plymouth, UK) and the London Hyperbaric and Wound Healing Centre (London, UK) between January 2004 and December 2010 to identify those cases presenting with symptoms suggestive of IEDCS. Divers presenting with symptoms of vestibulocochlear dysfunction (vertigo, nausea and vomiting, ataxia, hearing loss) occurring within 2 h of surfacing and in the absence of a history or signs of ear barotrauma were selected for inclusion in the study. This study was exempt from any ethical consideration as subjects were not treated as part of a research protocol and the audit was conducted on patients who were treated according to clinical need. Data is reported anonymously.

The following parameters were recorded from the medical notes of each subject: age, diving experience, depth of dive leading to incident, risk factors for decompression sickness (length of dive, rapid ascent, missed decompression stops, dehydration, extreme tiredness), breathing gas mixture used, time from surfacing to manifestation of symptoms, distribution of symptoms, cochlear involvement (hearing loss), time to first therapy, treatment table, number of treatments needed until no further improvement, initial recovery, recovery at 3-mo follow-up, past history of DCS, and presence of a RLS. All divers had been examined by diving physicians following the Institute of Naval Medicine Consultation Examination and Treatment Record. The neurological examination included a sharpened Romberg test, Unterberger test, cranial nerves examination, and cerebellar exam. Otoscopy had been performed on all patients.

Testing for a RLS

All divers treated for IEDCS were advised to be tested for the presence of a RLS. The presence of a RLS is assessed with bubble contrast transthoracic echocardiography, which is performed by a specialist cardiologist. In brief: a mixture of 8 ml saline, 1-2 ml withdrawn blood, and 1 ml of air are forced through a three-way tap between two 10-ml Leuer lock syringes to generate a contrast medium of microbubbles. This is injected into an antecubital fossa peripheral vein and the patient is asked to perform a Valsalva manoever for up to 20 s to reduce the venous return to the heart. Following release of the Valsalva, venous blood containing the contrast suddenly returns to the right side of the heart at a time when the left atrial pressure remains low. If there is a patent foramen ovale (PFO) or atrial septal defect (ASD), then bubbles can be seen entering the left heart at this point. The size of the shunt is described as minor, moderate, or severe depending on the number of bubbles seen in a still frame of the echo image when the maximum number of bubbles appear to be present and on whether there are any bubbles passing at rest. Current protocol is recommended by the UK sports diving medical committee and was described by Peter Wilmshurst (19).

Statistical Analysis

Statistics were performed with BIAS for Windows (Epsilon Verlag, Frankfurt, Germany; Version 9.14; 08/2011). Nominal data (recovery, no recovery with U.S. Navy table versus Comex table) was analyzed with the Fisher exact test, continuous data (number of treatments performed) was analyzed using the Wilcoxon-Mann-Whitney test because the data was not parametric. A statistical significant difference was set at $P < 0.05$.

RESULTS

Between January 2004 and December 2010 a total of 33 divers were treated for IEDCS. All had been diving in UK coastal waters in relatively cold water conditions using a dry suit. Of the 33 divers presenting with symptoms of vestibulo-cochlear dysfunction, 2 were women and 31 were men. Subjects were between the ages of 31-61 yr (mean 46) and had performed between 20-4000 dives (median 300). The maximum depth of the dive leading to the incident varied between 49 and 256 ft (median 103.7 ft) (15 and 78 m; median 31.6 m) with a median bottom time of 43 min. Of the divers, 22 had used air as a breathing gas (67%), 5 had used nitrox (15%), and 6 had used trimix (18%). Of the trimix divers, five were diving with a rebreather with constant partial pressure of oxygen and without gas switching. (**Table I**) Of the 33 divers, 16 experienced isolated IEDCS (48%) and 17 (52%) suffered associated symptoms ranging from pain in the joints, tingling, and sensation loss to paraparesis. One diver had an associated skin bend and another diver had brisk reflexes and sensation loss in glove and stocking fashion, which was attributed to excess consumption of alcohol and not considered an associated DCS symptom.

Of the risk factors investigated the most common were decompression diving and suspected dehydration, with i.v. fluids being administered to 27/33 subjects and a high proportion admitting to being dehydrated. Of interest to note is that for six divers it was a recurrent episode of DCS. Two suffered a previous episode of IEDCS, two previous neurological DCS, one has had recurrent skin bends, and one had pulmonary DCS. In all

TABLE I. SUMMARY OF SOME OF THE PARAMETERS ANALYZED.

Patient	Age	Gender	Max. Depth	Breathing Gas	Time to Onset of Symptoms (min)	Predisposing Factors	Further DCI Symptoms	Time to First Treatment (hours)	No of Treatments	Type of Initial Treatment	i.v. Fluids	R/L Shunt	Recovery at Discharge	Recovery 3 mo
1	46	M	36	Air	5	Missed deco, deco dive, lifting	No	7	14	USN TT6	Yes	PFO	No	No
2	52	M	30.5	Air	60	Dehydration, alcohol	No	1	9	USNTT6	Yes	PFO	No	Yes
3	52	M	24	Air	15	Dehydration, rapid ascent	No	2	9	Comex 30	Yes	PFO	No	Yes
4	39	M	21	Air	60	Dehydration, lifting	No	4	7	USN TT6	No	PFO	No	No
5	51	M	30.3	Nitrox	60	None	Yes	4.5	5	Comex 30	Yes	PFO	No	No
6	60	M	53.6	Air	10	Dehydration, deco dive, fast ascent	Yes	3.5	17	Comex 30	Yes	Negative	No	Yes
7	44	M	21	Air	0	Dehydration, skin bends previous	Yes	4	11	USN TT6	Yes	PFO	No	Yes
8	38	M	33	Air	10	Alcohol	Yes	3	9	Comex 30	Yes	ASD	No	Yes
9	31	F	44	Air	5	Alcohol	Yes	2.5	22	USN TT5	Yes	PFO	No	No
10	39	M	26	Air	120	Flu week before, alcohol	Yes	3.5	17	USN TT6	Yes	PFO	No	Yes
11	36	M	20	Air	60	Alcohol	Yes	2	16	USNTT6	Yes	PFO	No	Yes
12	33	M	64	Trimix/R	10	Dehydration, DCI previously	Yes	3.5	7	USN TT6	Yes	PFO	No	Yes
13	31	M	77	Trimix/R	10	Deco dive, dehydration, cough	Yes	3	6	Comex 30	Yes	PFO	No	No
14	46	M	65	Trimix/R	45	Deco dive, obese	Yes	2.5	6	Comex 30	Yes	PFO	No	Yes
15	49	M	78	Trimix	0	Deco dive, gas mix switch	Yes	5	14	USN TT6	Yes	Negative	No	Yes
16	37	M	22	Air	15	Dehydration	Yes	2.5	20	USN TT6	Yes	PFO	No	No
17	54	M	30.5	Air	30	Overweight, pulled boat, alcohol	No	4.5	9	Comex 30	Yes	PFO	No	Yes
18	50	M	46	Air	120	Deco dive, previous IEDCS	No	3	1	USN TT6	No	PFO	Yes	Yes
19	52	M	25	Air	30	Alcohol, previous neurol DCS	No	3	12	USN TT6	Yes	PFO	No	Yes
20	42	M	34.9	Air	60	Deco dive	No	4	1	USN TT6	No	PFO	Yes	Yes
21	40	M	60	Trimix/R	0	Alcohol	No	31	11	USN TT6	No	Negative	No	No
22	53	M	15	Air	70	None	Yes	10	7	USN TT6*	No	PFO	No	No
23	60	M	28	Air	20	Dehydration, excess alcohol	No	3	10	Comex 30	Yes	Refused	No	Yes
24	55	M	31.4	Air	5	Rapid ascent, missed stops	No	3	5	USN TT6	No	Negative	Yes	Yes
25	35	F	27.7	Air	5	Alcohol, dehydration	No	7	13	USN TT6	Yes	Negative	No	Yes
26	61	M	17	Air	30	Alcohol, dehydration	Yes	8	3	Comex 30	Yes	Refused	Yes	Yes
27	53	M	31.8	Air	30	Alcohol	Yes	3	7	Comex 30	Yes	Negative	Yes	Yes

TABLE I. CONTINUED

Patient	Age	Gender	Max. Depth	Breathing Gas	Time to Onset of Symptoms (min)	Predisposing Factors	Further DCI Symptoms	Time to First Treatment (hours)	No of Treatments	Type of Initial Treatment	i.v. Fluids	R/L Shunt	Recovery at Discharge	Recovery 3 mo
28	53	M	53	Trimix/R	60	Previous IEDCS	No	2.5	7	Comex 30	Yes	Refused	Yes	Yes
29	50	M	22	Air	0	Dehydration	No	12	10	USN TT6	Yes	PFO	No	No
30	43	M	46	Nitrox	0	Dehydration, deco dive	No	36	7	USN TT6	Yes	PFO	No	Yes
31	45	M	35	Nitrox	20	Deco dive	No	5	8	USN TT6	Yes	PFO	Yes	Yes
32	43	M	44	Nitrox	20	Deco dive	Yes	1	4	USN TT6	Yes	PFO	No	Yes
33	37	M	32	Nitrox	10	Deco dive	No	4	8	USN TT6	Yes	PFO	No	No

Trimix/R = rebreather; alcohol = drinking the night before; dehydration = patient saying fluid intake low.
USN TT6 = U.S. Navy Treatment Table 6; Comex 30 = Companie Maritime d'Expertise; USN TT5 = U.S. Navy Treatment Table 5; R/L shunt = right-to-left shunt; PFO = patent foramen ovale; ASD = atrial septal defect.

but one patient the main symptoms described was vertigo. Of the 33 patients only 20 received a documented hearing test and of these 13 exhibited hearing loss in the high frequencies. Only one patient complained of hearing loss (pancochlear) as an isolated symptom.

The median time between onset of symptoms and recompression therapy was 3.5 h (range 1-36 h). There was no indication that time to treatment affected speed of recovery. In 21 patients the initial recompression table used was a U.S. Navy Treatment Table 6 (USN TT6) and in 11 cases a Companie Maritime d'Expertise (Comex 30; now Stolt-Offshore) was used. One patient was initially treated with a U.S. Navy Treatment Table 5 (USN TT5) and retreated the next day using a Comex 30 table after developing spinal cord DCI with a level at T10 and was not completely recovered at discharge or at follow-up. Another patient was initially treated at another chamber using a USN TT6 which was aborted due to compressor failure. The patient was recompressed on a USN TT6 the following morning. No significant difference could be found regarding the treatment table used in terms of absolute recovery. Three divers treated with the Comex 30 table made a full initial recovery whereas eight did not compared to four divers treated with USN TT6 with full initial recovery contrasted with 17 without recovery **(**P = 0.66**).** There was also no statistical significant difference in the 3 mo follow-up recovery between both tables. Of 11 divers who were treated with a Comex 30 table, 9 made a full recovery at the 3-mo follow-up and 2 did not compared to 21 divers treated using a USN TT6 with 14 divers who made a full recovery at follow-up and 7 divers without full recovery at follow-up **(**P = 0.44**).**

Of the 32 patients receiving an initial treatment table of either a USN TT6 or a Comex 30 there was no difference in the number of treatments needed to recover or reach plateau (which means no further recovery noticed after treatment). Patients treated with a Comex 30 table needed a median of 7 treatments (range 3–17) until no improvement was noted, whereas divers treated with USN TT6 needed a median of 9 treatments (range 1–20) until no improvement of symptoms was found. There was no statistically significant difference between both groups **(**P = 0.62**)**. Please note that only 32 patients were included in the statistics comparing the USN TT6 with Comex 30 due to the fact that 1 patient was initially treated with a USN TT5. In the complete series of all 33 divers, 1–22 treatments were required to reach plateau (median = 9). There was also no correlation found between delay of treatment and number of treatments needed (P = 0.79). A graphical analysis and a regression analysis (Pearson) were performed.

All 33 divers were advised to undergo bubble contrast echocardiography to investigate the potential presence of a RLS. Of the 33 divers only 30 underwent the test, with 1 patient deciding to cease diving after the incident and 2 divers electing not to undergo the test. Of the 30 divers who underwent the test, 24 tested positive for a RLS (80%), with 23 having a severe PFO and 1 having an ASD. Only seven patients (21%) had recovered at discharge from the chamber. At the 3-mo follow-up, 23

divers had recovered and 10 divers had not recovered completely. Of the 10 divers that had not recovered after the 3 mo, 7 had persistent inner ear problems (episodes of vertigo, hearing loss) and the remaining 3 had other persistent neurology such as memory problems and spinal cord deficit (motor and sensory deficit).

DISCUSSION

Until last decade IEDCS was considered to be a rare condition in sport scuba divers and predominantly linked to the switching of gases during decompression—a phenomenon described as 'inert gas counter-diffusion'. Farmer was one of the first to report 23 cases of hearing loss, tinnitus, and vertigo occurring during or shortly after decompression. Of these cases, 13 (57%) occurred after helium-oxygen dives involving a change to air during the latter stages of decompression (5).

Recent case series by Nachum, Cantais, and Klingmann have highlighted the increased awareness of IEDCS in recreational divers diving with air or nitrox (3,9,12). In the case series presented here, 33 divers (from a total of 662 patients treated for DCI) were diagnosed as having suffered IEDCS, which represents an overall incidence of 4.9% of all cases treated. Other studies of incidences of IEDCS have presented their data as a proportion of neurological DCS rather than total DCI and reported incidence rates of 26% and 33%, respectively (3,12). Subgroup analysis of data included in this study from the Diving Diseases Research Centre demonstrates the incidence of IEDCS as a proportion of neurological DCS to be 12.5% (28 from 223), which is significant but lower than that reported in other studies.

Of the 33 divers in our series, 22 had used air as a breathing gas (67%), 5 divers had used nitrox (15%), and 6 had used trimix (18%). One patient in the trimix group had symptoms starting under water at 29.5 ft (9 m), which might have been due to an incorrect gas switch at 98 ft (30 m) as his buddy found him confused and had to help him to the surface. Analyzing the incidence of IEDCS in the 3 separate groups of divers (air, nitrox, and trimix), we noted that from a subgroup of 382 records analyzed (patients treated at DDRC), 303 divers had used air, 51 had dived with trimix, and 28 with nitrox. The incidence of IEDCS was 6.9% (21 divers) in the air group, 11.7% (6 divers) in the trimix group, and 3.5% (1diver) in the nitrox group.

The most common risk factors encountered in our series was dehydration (often linked to alcohol consumption and decompression dives). Whereas a majority of patients presented with a history of one or two episodes of vomiting, this was rarely sufficiently severe to attribute the need for fluids. In general the cause of dehydration was attributed to primary dehydration and not loss of fluids through vomiting.

Of the 33 IEDCS patients, only 20 underwent a documented hearing test and of those 13 exhibited hearing loss at high frequencies. This is similar to a previous study that found a hearing loss in 40% of their cases (7). Only one of our patients had additional steroid and/or rheological therapy as described by Klingmann (9). Only one patient presented with hearing loss as an isolated symptom and this was the patient treated with high-dose steroids in addition to the hyperbaric treatment. The occurrence of isolated cochlear dysfunction as a symptom of IEDCS has been previously reported by Nachum and Klingmann (9,12).

There were 16 patients who demonstrated isolated IEDCS (48%) and 17 (52%) who had associated symptoms ranging from heaviness in the joints, tingling, and sensation loss to paraparesis. In other published series, isolated IEDCS was found in 52% and 83% of the divers, respectively (7,12). In the inner ear the labyrinthine artery (auditory artery, internal auditory artery) is a long slender branch of the anterior inferior cerebellar artery (85–100% cases) or basilar artery (< 15% cases) which accompanies the vestibulocochlear nerve through the internal acoustic meatus and is distributed to the internal ear. It divides into two cochleo-vestibular arteries—the anterior vestibular artery and the vestibular-cochlear artery. The anterior vestibular artery supplies the lateral and anterior ampullae, and the vestibular-cochlear artery supplies the posterior ampulla and the cochlea (16). The basilar artery also supplies the brainstem and branches into the superior cerebellar artery and the posterior cerebral artery. This anatomy dictates that if bubbles enter the labyrinthine artery they must also distribute to the arteries supplying the brainstem and cerebellum. Yet in isolated cases of pure peripheral vestibulo-cochlear deficit we do not see signs of an embolic event in this area.

One possible explanation for the increased susceptibility of the inner ear for isolated IEDCS has been offered by Mitchell and Doolette (11). They have described a theoretical model to explain why the inner ear may be more susceptible to decompression sickness as compared to the brain. The model is based on previous studies of the same authors on inert gas kinetics and the ability to predict gas supersaturation conditions (4). Supersaturation refers to the sum of tissue gas partial pressures being greater than ambient pressure, a condition necessary for bubble growth (17). Their model predicts a half-life for nitrogen washout in the inner ear of 8.8 min and 1.2 min for the brain. Using this model they demonstrated that after a dive the inner ear remains supersaturated with inert gas for longer than brain tissues, and the window of opportunity for growth of small vascular bubbles distributing to the labyrinthine artery territory is correspondingly longer (11). Thus, if a RLS is present, bubbles may pass from the venous to arterial circulation and arrive in the inner ear while it remains supersaturated with inert gas. These bubbles tend to grow as the supersaturated gas diffuses into them, whereas bubbles arriving in the cerebral circulation will not grow because the surrounding tissue is not supersaturated.

At least three conditions seem necessary in order to support the hypothesis of an arterial embolization:

1. The existence of a RLS, preferably permanent and severe, where the origin is most often cardiac;
2. The presence of a sufficient circulating venous bubble load; and
3. The presence of a pressure gradient capable of pushing these bubbles from the right to the left side of the heart via a shunt.

These three conditions can be present during and after a dive and thus make an arterial embolization plausible (18). In our study all three conditions were potentially present: we found a RLS shunt in 80% of the divers, which is in concordance with previous reports (3,9). There were 23 divers who demonstrated a severe PFO and 1 had an ASD, and all dives were decompression dives or high bubble load dives. Regarding the third condition (pressure gradient) we can only speculate, but some of the divers reported onset of symptoms after coughing, lifting heavy gear, and lifting themselves into the diving boat (these maneuvers would increase the pressure in the right side of the heart).

The explanation described above does not explain IEDCS in the 20% of the divers that were PFO negative and where other mechanisms must be considered. One explanation could be that local bubble growth in absence of arterial bubbles occurs in the inner ear (4). Further research is necessary to explain these cases.

The median time between occurrence of symptoms and first hyperbaric oxygen therapy was 3.5 h with a range of 1-36 h. (2 divers, 5.8%, were treated within 1 h). There was no difference in number of treatments needed if patients were treated earlier rather than later as assessed by balance tests, persistence of nystagmus, and subjective reports of the divers. In previous studies it has been reported that significant improvement of the symptoms is observed only when recompression is commenced within 68 min of appearance of symptoms (5). Long-term observation of patients in the group of Shupak et al. demonstrated that when the median latency period before recompression was 5 h long-term damage resulted in 90% of divers with IEDCS (15). In our study 26 of the 33 patients (78.7%) were treated within 5 h of onset of symptoms and our median latency period was 3.5 h.

One shortcoming of this study in addition to it being a retrospective review is that none of the subjects received an ENT follow-up with appropriate evaluation. Whether the patients had recovered or not was based on balance tests, audiometry, and subjective symptoms of the patients. Klingmann et al. reported a residual cochleo-vestibular deficit in 78% of patients, but his study included only patients who presented to the ENT department after the incident and it follows that patients who still have symptoms will be those seeking specialist advice. In his series only 70% of the patients with IEDCS had received hyperbaric oxygen treatment (9).

In accordance with previous studies, only seven patients (21%) had recovered at discharge (12). At the 3-mo follow-up, 23 divers had recovered and 7 of the 10 that had not recovered completely had vestibular deficits. Long-term follow-up of 1 yr and longer showed though that all but two patients had recovered from a vestibular aspect. The two patients that had not recovered had cochlear deficit only. As these patients did not have an appropriate ENT examination at follow-up, it is difficult to determine the exact nature of the residual vestibulocohlear deficit. Shupak et al. have previously reported in their series of 11 divers with IEDCS and 9 divers with inner ear barotrauma that 8 patients had residual vestibular deficit (40%) at follow-up, but only 1 was symptomatic (15). We, therefore, speculate that although all our patients were asymptomatic at the long-term follow-up, in absence of proper vestibular testing methods (electronystagmography, rotator chair test using the sinusoidal harmonic acceleration protocol, computerized dynamic posturography or energy video-oculography), the percentage of real recovery of vestibulo-cohlear function was lower (9,15).

It is interesting to note that 32/33 divers in our series suffered with vestibular symptoms with or without cochlear involvement and only 1 diver had isolated cochlear dysfunction. There was no functional anastomosis of blood vessels between the middle and the inner ear, despite their close anatomical relationship (13). From studies in rabbits it is known that vestibular blood flow is three times lower than the cochlear blood flow (1). Nakashima et al. showed that the blood flow to the vestibular system of guinea pigs was even four times lower than blood flow to the cochlea (14). One possible explanation of the increased susceptibility of the vestibular organ to DCS could be linked to the observation in our study that over 50% of the divers treated were dehydrated (needed i.v fluids to produce urinary output and admitted to low fluid intake preceding the dive). It would, therefore, be plausible to speculate that hypovolaemia would further reduce blood flow in the vestibular organ as well as altering the rheology, causing embolization and platelet aggregation, also explaining the unsatisfactory response of IEDCS to recompression therapy. This is only a speculation though, as diving is generally associated with different grades of dehydration. Boussuges et al. have shown that a hematocrit level $\geq 48\%$ was correlated with persistent neurological sequelae 1 mo after the accident in a study of 58 recreational divers treated for neurological decompression sickness (2). It is even more likely though, that due to the lower blood flow in the vestibular system, the half-time of desaturation in the vestibular part of the labyrinth is lower than in the cochlea and, therefore, the vestibular organ is more prone to bubble growth than the cochlea.

Our study is the most complete retrospective analysis describing variables not reported in previous accounts (type of recompression table used, number of treatments) concerning IEDCS. Our data confirms the high prevalence of vestibular symptoms compared to cochlear dysfunction in IEDCS and attempts to explain this finding. It also confirms the correlation between IEDCS and presence of a significant PFO. In our series 48% of patients had an isolated IEDCS for which both the combined presence of a RLS and gas supersaturation state in the inner ear could be responsible. There are, however, a small percentage of divers exhibiting signs and symptoms of IEDCS that are PFO negative and, for these patients, another mechanism has to be considered. Our study also demonstrates that IEDCS responds slowly to treatment irrespective of the initial table used. The recovery is thought to be mainly a central compensation process.

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Author Query sheet–ASEM3326

Q1 : Author: We normally provide both feet and meters for depth and altitude. Please check that the conversions are acceptable.
Q2 : Author: These two sentences (“This study was exempt … reported anonymously.”) were moved here from the ‘Statistical Analysis’ section as they seem to fit better here. Is this OK?