# Original articles

## A descriptive epidemiological analysis of isolated inner ear decompression illness in recreational divers in Hawaii

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**Key words**
Decompression illness,decompression sickness. inner ear decompression illness, inner ear decompression sickness, treatment, epidemiology

**Abstract**

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Inner ear decompression illness (IEDCI) was once thought to be relatively rare and seen predominantly in deep, mixed-gas divers. The incidence of this type of injury is unknown, but IEDCI may be more common than previously thought and can be seen in recreational scuba divers using compressed air as their breathing medium. This study was conducted at the Hyperbaric Treatment Center (HTC) in Honolulu, Hawaii, to determine the frequency of occurrence of IEDCI and to evaluate some of the epidemiological parameters associated with these cases. Between 1983 and 2006, 28 presentations (2.8% of all cases of decompression illness treated) with a diagnosis of isolated IEDCI were identified in 26 divers. Presenting symptoms and physical findings included vertigo, nausea, postural imbalance, vomiting, nystagmus, hearing loss, and tinnitus. Most cases developed after multiple deep dives or after dives in which adequate decompression did not occur. All but two divers were breathing air. Symptoms developed on average 70 minutes after diving. The average delay to treatment was nine hours post injury. All but three cases were treated using the HTC deep treatment tables. Nineteen cases made a full recovery, with all cases achieving substantial improvement. Most cases required four to five treatments to obtain that level of recovery. Those with incomplete resolution of symptoms at the time of discharge were left with mild degrees of motion sickness and gait disturbance, and some were left with hearing loss. IEDCI warrants early and aggressive intervention to reduce the risk of permanent disability.

### Introduction

Isolated inner ear decompression illness (IEDCI) is an acute peripheral cochleo-vestibular disturbance arising as a consequence of breathing compressed gas at increased atmospheric pressures. It is characterised by symptoms and physical findings of acute vertigo, nausea and postural imbalance, and perhaps vomiting, hearing loss, and tinnitus. It has historically been reported rarely. AH Smith may have been the first to describe the symptoms of what is now referred to as inner ear decompression sickness (IEDCS) in caisson workers when he noted both deafness and vestibular problems in compressed air workers in 1873.[1] Later, in 1929, Vail demonstrated that inner ear damage could occur during decompression from embolisation of nitrogen bubbles and result in necrosis of those tissues.[2] For a period of time thereafter, IEDCI was infrequently noted. The frequency of cases may have been lessened through the implementation of improved safety procedures, but this is also because IEDCI was often seen only in cases of more extensive neurological injury and thought to be part of a larger syndrome. Thus, IEDCI was relegated to a finding of lesser import and not regarded as a separate entity.[3]

The incidence of IEDCI in any diving population, be it commercial, scientific, military or recreational, is unknown. It had been thought to occur largely in deep, mixed-gas divers or saturation divers in commercial diving. The symptoms of IEDCI may accompany inner ear barotrauma or be part of a more global presentation of decompression sickness (DCS) affecting the brain itself, which can make the diagnosis of IEDCI rather challenging for the diving physician.[4] In the 1970s, several reports again focused attention on the possibility that IEDCI might also be a discrete finding.[5–8] It was not reported in recreational divers using compressed air until the 1990s.[9–11] More recently, several studies appear to associate isolated IEDCI with the existence of a patent foramen ovale (PFO).[12–14]

This study reports on the experiences at the Hyperbaric Treatment Center (HTC) of the University of Hawaii John A Burns School of Medicine in evaluating and treating isolated IEDCI, that is 'stand-alone' cases of IEDCI, in recreational divers.

### Methods

Characterisation of this clinical syndrome with the more general term IEDCI vis-à-vis IEDCS is used except where IEDCS is specifically meant. A review of 1,422 patient records was undertaken by the author to determine the number of isolated IEDCI cases treated at the HTC between

the years 1983 and 2006. The study was reviewed by the Committee on Human Studies of the University of Hawaii and determined to be exempt from the Department of Health and Human Services regulations, 45CFR Part 46. All possible cases were considered for inclusion in this study. A possible case was defined as one who had been diagnosed with IEDCI in the HTC database at the time of treatment, as well as any diver who had presented with symptoms of at least vertigo, nausea, and/or vomiting, and might also have had ataxia, tinnitus and/or hearing loss. Confirmed and/or probable cases were defined as those who evinced only a peripheral cochleo-vestibular abnormality resulting from decompression stress or arterial embolism.

Confirmed and probable cases were determined after a complete review of the clinical records of each identified possible case based upon the recorded past medical history, history of presentation, diving history of the incident event(s), physical examinations conducted, and any neurological and/or otological assessments performed at the time of treatment. Divers who presented with histories and physical findings consistent with a more extensive expression of DCS, inner ear barotrauma, viral labyrinthitis, vestibular neuronitis, Ménière's disease, vertebral and basilar artery disease or cerebellar disease were excluded.[15,16]

Epidemiological data focusing on gender, age, dive profile, breathing gas employed, time to onset of symptoms, time delay to treatment, presenting symptoms, physical findings, and HTC treatment regimen undertaken for confirmed or probable cases were extracted and analysed. Outcome of treatment was assessed by comparing pre- and post-treatment severity scores devised by the HTC and used to determine efficacy and outcomes of the HTC treatment tables in a previous study.[17] Since the goal of treatment is to restore the patient to as near a state of normal functionality as possible, the ability to conduct routine, normal activities of daily living (ADL) was chosen as the outcome measure. ADL were defined as the routine, unencumbered physical and mental functions normally undertaken by the patient prior to the diving injury. During the chart review, each patient was assigned a pre-treatment initial functional impairment score (iFIS) of one to four based upon physical findings at the time of presentation and impact on ability to conduct routine ADL (Table 1).

For iFIS scoring in general, minor subjective symptoms included dizziness, motion sickness, heaviness, malaise, lightheadedness, headache and fatigue. Mild to moderate symptoms/signs included minor balance problems, minor weakness or loss of sensation, aches, tingling and numbness. Substantive symptoms/signs included incapacitating vertigo, major disturbance of balance, hearing loss, paresis, paralysis, paraplegia, bowel/bladder dysfunction, altered mental status, altered vision and incapacitating pain. Life-threatening symptoms/signs included cardiopulmonary arrest and severe central neurological injury.

**Table 1**
**Honolulu Hyperbaric Treatment Center functional impairment scoring system (ADL – activities of daily living)**

| Score | Definition of level of impairment |
|---|---|
| 0* | No physical signs/symptoms, no impairment/limitations to ADL |
| 1 | Minor subjective symptoms, no physical signs, no impact/limitations to ADL |
| 2 | Moderate objective signs/symptoms, mild to moderate impact/limitations to ADL |
| 3 | Major objective signs/symptoms, substantial impact/limitations to ADL |
| 4 | Life-threatening signs/symptoms, severe immediate impact/limitations to ADL |

*Used for post-treatment scoring only

Patients were assigned a residual functional impairment score (rFIS) of zero to four at the time of discharge from care at the HTC using the same scoring system as for the iFIS (Table 1). Pre- and post-treatment scores (iFIS and rFIS) were compared to determine improvement.[17]

The treatment tables employed to treat these cases were those routinely used at the HTC and are briefly summarised here.

TT60

This begins with recompression to 60 feet sea water (fsw, 18 metres' sea water (msw); 283 kPa) where the patient is placed on 100% oxygen ($O_2$). Three $O_2$ periods are undertaken at 18 msw, then three at 13.6 msw, two at 9 msw, and two at 4.5 msw, then ascent to the surface. All $O_2$ periods are 20 minutes in length interspersed with 5-minute air breaks. Up to three $O_2$ period extensions may be used at 18 msw and/or 13.6 msw; peak $ppO_2$ at 18 msw = 284 kPa.

TT160

This begins with a deep compression to 160 fsw (48.4 msw; 588 kPa) on air. Upon reaching 48.4 msw, the patient is placed on a 50/50 $N_2/O_2$ gas mix for 30 minutes, followed by a slow staged decompression to 18 msw where the gas is changed to 100% $O_2$. The schedule thereafter is as for TT60; peak $ppO_2$ at 48.4 msw = 294 kPa; at 18 msw = 284 kPa.

TT220

This begins with deep compression to 220 fsw (66.6 msw; 774 kPa) on air. Upon reaching 66.6 msw, the patient is placed on a 65/35 $N_2/O_2$ gas mix for 15 minutes, followed by a slow staged decompression to 45.4 msw where the gas mix is changed to 50/50 with a still slower staged ascent to

**Table 2. Dive, symptom and treatment characteristics of 28 cases (all divers but 11 and 13 were diving on air; numbers in parentheses after dives represent**

| Diver | Dive profiles | Symptoms | Onset* (min) | Delay† (min) | Physical findings | iFIS | Treatment regimen | rFIS |
|---|---|---|---|---|---|---|---|---|
| 1 | 26 msw x 50 min<br>1:30 hr SI<br>9 msw x 50 min | Vt, N | 300 | 1,320 | Nystagmus (h,L)<br>PI (R) | 3 | 774 kPa x 3<br>284 kPa x 1<br>ENT eval [A,E] | 1 |
| 2 | 50 msw x 8 min<br>3:00 hr SI<br>38 msw x 40 min | Vt, N | 30 | 360 | Nystagmus (h,L)<br>PI (R)<br>Hearing loss (s/n,R) | 3 | 774 kPa x 2<br>284 kPa x 4<br>ENT/Neuro eval [A,Tp,E] | 0 |
| 3 | 26 msw x 60 min | Vt, N, V | 20 | 420 | Nystagmus (h,L)<br>PI (R) | 3 | 774 kPa x 2<br>588 kPa x 1<br>284 kPa x 4 | 0 |
| 4 | 27 msw x 30 min (2)<br>0:45 hr SI<br>27 msw x 30 min | Vt, N, V, HL | 30 | 240 | Nystagmus (h,R)<br>PI (L)<br>Hearing loss (s/n,L) | 3 | 774 kPa x 6<br>ENT eval [A,Tp,E] | 0 |
| 5 | 23 msw x 56 min<br>1:10 hr SI<br>18 msw x 62 min | Vt, N, V | 60 | 500 | Nystagmus (h,L)<br>PI (R) | 2 | 774 kPa x 2 | 0 |
| 6 | 27 msw x 30 min (4) | Vt, N, V | 10 | 165 | Nystagmus (h,R)<br>PI (L) | 3 | 774 kPa x 1<br>588 kPa x 2<br>284 kPa x 3<br>ENT eval [A,Tp,E] | 0 |
| 7 | 27 msw x 30 min (2)<br>18 msw x 40 min (2)<br>SIs < 10 min | Vt, N, V | 30 | 180 | Nystagmus (h,R)<br>PI (L) | 3 | 774 kPa x 4<br>284 kPa x 3<br>ENT eval [A,Tp,E] | 0 |
| 8 | 30 msw x 25 min (2)<br>26 msw x 25 min (2)<br>15 msw x 25 min (2)<br>SIs < 10 min | Vt, N, V | 25 | 180 | Nystagmus (h,R)<br>PI (L) | 3 | 774 kPa x 2<br>284 kPa x 5 | 1 |
| 9 | 35 msw x 15 min<br>1:15 hr SI<br>27 msw x 25 min | Vt, N, V, HL | 20 | 360 | Nystagmus (h,L)<br>PI (R)<br>Hearing loss (s/n,R) | 3 | 774 kPa x 2<br>284 kPa x 5<br>ENT/Neuro eval [A,Tp,E] | 1 |
| 10 | 20 msw x 60 min<br>1:00 hr SI<br>18 msw x 50 min | Vt, N, V | 960 | 480 | Nystagmus (h,L)<br>PI (R) | 2 | 774 kPa x 1<br>284 kPa x 3<br>ENT eval [A,E] | 0 |
| 11 | 54 msw x 180 min<br>Deco time 105 min | Vt, N, V | 100 | 430 | Nystagmus (h,R)<br>PI (L) | 3 | 774 kPa x 2 | 0 |
| 12 | 27 msw x 44 min | Vt | 15 | 420 | Nystagmus (h,L)<br>PI (R) | 3 | 774 kPa x 3<br>284 kPa x 1 | 1 |
| 13 | 60 msw x 74 min<br>Deco time 28 min | Vt, N, V | 120 | 450 | Nystagmus (h,L) | 2 | 284 kPa x 1 | 0 |
| 14 | 56 msw x 26 min<br>Deco stops: ad hoc<br>Rapid ascent<br>IWR@ 6 msw x 20 min (37% nitrox) | Vt, N, V | 10 | 720 | PI (R) | 2 | 774 kPa x 1 | 0 |

*time to onset of symptoms post dive; †delay from onset of symptoms to treatment
iFIS – initial functional impairment score; rFIS – residual functional impairment score
**Dive profiles:** SI – surface interval; IWR – in-water recompression
**Symptoms:** Vt – vertigo; N – nausea; V – vomiting; HL – hearing loss; T – tinnitus
**Physical findings:** h – horizontal; t – torsional; R – right; L – left; U – upper; s/n – sensorineural; PI – postural imbalance
**Investigations:** A – audiogram, Tp – tympanogram, E – electronystagmogram

**of confirmed or probable isolated inner ear decompression illness**
**the number of dives; if decompression stops were done, the total stop times are shown)**

| Diver | Dive profiles | Symptoms | Onset* (min) | Delay† (min) | Physical findings | iFIS | Treatment regimen | rFIS |
|---|---|---|---|---|---|---|---|---|
| 15 | 15 msw x 40 min | Vt, N, V | 10 | 360 | Nystagmus (h,R)<br>PI (L) | 3 | 774 kPa x 1<br>588 kPa x 1<br>284 kPa x 4<br>ENT/Neuro eval [A,E] | 0 |
| 16 | 60 msw x 30 min | Vt, N, V, HL, T | 10 | 360 | Nystagmus (h,L)<br>PI (R)<br>Hearing loss (s/n,R) | 3 | 774 kPa x 1<br>588 kPa x 1<br>284 kPa x 4<br>ENT eval [A,Tp,E] | 1 |
| 17 | 27 msw x 40 min | Vt, N, V, HL | 30 | 140 | Nystagmus (h,R)<br>PI (L)<br>Hearing loss (s/n,L) | 3 | 774 kPa x 1<br>243 kPa x 25<br>ENT eval [A,E] | 1 |
| 18 | 33 msw x 30 min (2)<br>24 msw x 30 min (2)<br>18 msw x 40 min<br>21 msw x 30 min<br>SIs < 10 min | Vt, N, V, HL | 60 | 720 | Nystagmus (t,U)<br>PI (L)<br>Hearing loss (s/n,L) | 3 | 284 kPa x 2<br>243 kPa x 3<br>ENT/Neuro eval [A,Tp,E] | 1 |
| 19 | 33 msw x 15 min (2)<br>39 msw x 10 min<br>23 msw x 40 min (4)<br>SIs < 10 min<br>Deco time (last dive) 12 min | Vt, N, V | 30 | 360 | Nystagmus (h,L)<br>PI (R) | 3 | 588 kPa x 2<br>ENT eval [A,E] | 0 |
| 20 | 27 msw x 50 min<br>1:00 hr SI<br>18 msw x 50 min | Vt, N, V | 60 | 550 | Nystagmus (h,L)<br>PI (R) | 3 | 774 kPa x 1<br>284 kPa x 1<br>Echocardiogram | 0 |
| 21 | 48 msw x 48 min<br>Deco time 20 min<br>1:27 hr SI<br>43 msw x 56 min<br>Deco time 24 min | Vt, N, V | 30 | 360 | Nystagmus (h,L)<br>PI (R) | 3 | 774 kPa x 2<br>284 kPa x 1<br>ENT eval [A,E] | 0 |
| 22 | 33.5 msw x 24 min<br>0:35 hr SI<br>13.5 msw x 34 min | Vt, N, V | 10 | 480 | Nystagmus (h,R)<br>PI (L) | 2 | 774 kPa x 1<br>ENT eval [A,E] | 0 |
| 23 | 16.5 msw x 35 min<br>0:15 hr SI<br>15 msw x 45 min<br>23 msw x 35 min | Vt, N, V, HL, T | 20 | 2,110 | Nystagmus (h,L)<br>PI (R)<br>Hearing loss (s/n,R) | 2 | 284 kPa x 4<br>ENT eval [A,Tp,E] | 1 |
| 24 | 24 msw x 40 min<br>Deco time 4 min | Vt, N, V, HL | 15 | 180 | Nystagmus (h,R)<br>PI (L)<br>Hearing loss (s/n,L) | 3 | 774 kPa x 1<br>284 kPa x 1 | 0 |
| 25 | 27 msw x 30 min<br>Loss of buoyancy<br>Rapid ascent | Vt, N, HL | 10 | 1,440 | PI (L)<br>Hearing loss (s/n,L) | 2 | 774 kPa x 1 | 0 |
| 26 | 27 msw x 30 min | Vt, N, V | 30 | 140 | Nystagmus (h,R)<br>PI (L) | 3 | 588 kPa x 1<br>243 kPa x 16<br>ENT eval [A,Tp,E] | 1 |
| 27 | 21 msw x 30 min | Vt, N, V | 20 | 120 | Nystagmus (h,L)<br>PI (R) | 3 | 774 kPa x 1<br>242 kPa x 7<br>ENT eval [A,E] | 0 |
| 28 | 18 msw x 41 min<br>1:41 hr SI<br>12 msw x 52 min | Vt, HL | 10 | 1,440 | Nystagmus (h,L)<br>PI (R)<br>Hearing loss (s/n,R) | 3 | 774 kPa x 7<br>ENT/Neuro eval [A,E] | 0 |

18 msw where the gas is changed to 100% $O_2$. The schedule thereafter is as for TT60; peak $ppO_2$ at 66.6 msw = 271 kPa; at 45.4 msw = 281 kPa; at 18 msw = 284 kPa.

TT47

A few cases received follow-up treatments at 47 fsw (14 msw, 243 kPa), 100% $O_2$, four $O_2$ periods of 20 minutes each.

**Results**

A total of 61 possible cases presented with at least vertigo, nausea and/or vomiting or had been previously classified as IEDCI. Of these, 28 presentations in 26 divers were ultimately determined to be confirmed or probable cases of isolated IEDCI. Five cases originally classified as having isolated IEDCI were reclassified as having a more global DCS problem as a result of this review. The 28 cases of isolated IEDCI constituted 2.8% of all cases treated for decompression illness (DCI) at the HTC, an average case rate of 1.2 per year. All were male divers with an average age of 46 years (range 20–77 years).

The breathing gas was compressed air in 26 of the 28 cases, and trimix (helium, nitrogen, oxygen) in the other two cases. The average dive depth was 32.7 msw (108 fsw) with a range of 15–60.6 msw (50–200 fsw). Twenty-two cases made multiple deep dives in rapid succession (with surface intervals of less than 10 minutes) for extended periods of time, or single decompression dives without making decompression stops at all, or not achieving adequate decompression or exceeding no-decompression limits. Six cases developed symptoms after 'routine' dives to depths ranging from 15–27 msw (50–90 fsw) that were either at the edge of the no-decompression limits or within them. Three of these 'routine' dive cases appear to have embolised, which may explain their particular circumstances. The average time to onset of symptoms was about 70 minutes post dive (range 10–960 minutes, median 25–30 minutes).

The specific dive profiles for each case, as well as initial symptoms and their time to onset after diving are shown in Table 2. The dive profiles are listed in the sequence in which they occurred, with surface intervals, when undertaken, shown where they actually were effected. Only six divers (Divers 11, 13, 14, 19, 21 and 24), including the two mixed-gas divers, undertook decompression stops on any of their dives.

Divers 15, 25, and 28 were thought to have had arterial gas emboli from pulmonary barotrauma as the mechanism of injury leading to their specific symptoms and physical findings. Significant histories supporting this contention were, respectively, a breath-hold ascent, loss of buoyancy control with rapid ascent, and asthma with demonstrated air trapping on lung scan.

Initial reported symptoms in decreasing order of frequency were vertigo (28/28), nausea (26/28), vomiting (23/28), hearing loss (9/28), and tinnitus (2/28). The two cases who experienced tinnitus also suffered hearing loss. There were no cases in which hearing loss alone was a presenting problem.

Table 2 also depicts the delay to treatment time from onset of symptoms, the relevant physical findings, the iFIS, the treatment regimen to include any additional expert evaluations and the rFIS at the time of discharge. The average time delay to treatment was 9 hours (range 2–35 hours, median 6 hours).

On physical examination, 27 cases were found to have postural imbalance, 26 had nystagmus, 10 had hearing loss, and two complained of tinnitus. None of the 28 cases had a history of difficulty with equalisation during descent or ascent, ear blockage, or ear fullness, nor did they evince physical findings of middle ear barotrauma or tympanic immobility on physical exam. All were able to autoinflate their middle ears without difficulty.

The finding of nystagmus was accomplished using a combination of physical exam to include use of Frenzel lenses, and in some cases only after electronystagmography (ENG) was performed by an otologist. All except divers 14, 18, and 25 demonstrated horizontal nystagmus with the fast component in the direction as listed in Table 2. The finding of an upward, torsional nystagmus in diver 18 was confirmed by both an evaluating otologist and neurologist, both of whom believed the lesion was peripheral. Likewise in those cases where ENG was performed, peripheral vestibulopathy was confirmed. In divers 14 and 25, nystagmus was not clinically detected nor studied by ENG.

Postural imbalance was assessed based upon the inability of the patient to stand or ambulate without falling, and, if capable, on performance of Romberg's test, one-leg standing, Fukuda's test, Unterberger's test and heel-toe walking. More than half of these patients initially presented with prostrating vertigo and were able to be more fully tested only after the initial recompression treatment. Direction of leaning or falling on exam is recorded in Table 2. In all cases except divers 14 and 25, the direction of fall or leaning with postural imbalance was in the opposite direction to the fast phase of nystagmus as determined on examination or ENG.[16]

Hearing was routinely tested using standard tuning forks (Weber, Rinne, Schwabach tests), and in the 10 cases with hearing loss, sensorineural loss was suggested based upon findings of absent bone conduction in one ear (N = 8) and loss of both air and bone conduction in one ear (N = 2, with Rinne and lateralisation to the normal ear with Weber).[18] Interestingly diver 2 was found to have hearing loss even though he had not noted it as a complaint.

Eighteen (64%) cases, including eight of the 10 cases with hearing loss, were evaluated by an otologist at some point in their treatment regimen with testing via audiogram, ENG, and tympanography as deemed appropriate (Table 2). For those cases in which an audiogram was performed, a sensorineural hearing loss of 20 decibels or greater was observed (specifically reported in six of the eight cases as being in the mid- to high-frequency range).

Twenty-one cases had an iFIS of 3 on admission, indicating substantial limitations. The remaining seven cases had a moderate degree of impairment (iFIS = 2). Nineteen cases were discharged with an rFIS of 0, indicating full functional recovery, while nine were discharged with an rFIS of 1, indicating some mild residual symptoms principally manifest by a sense of motion sickness with rapid movement, a wide-based gait or some residual hearing loss. Five of the 10 cases who presented with hearing loss still had some hearing deficit at the time of discharge, though all had improved clinically. No post-discharge audiological evaluations were available for review.

The sequence of recompression treatments is shown in Table 2 in the order in which they occurred as well as the number of treatments. The average number of treatments required was 5 (range 1–26, median 4–5). All but three cases were initially treated on one of the HTC deep tables.[17] No specific rationale for why two of these three cases were not treated on a deep table could be ascertained from review of the records. Diver 13 was treated on the TT60 (284 kPa) because his symptoms had appreciably resolved prior to his arrival at the HTC. He was the only one of the three treated at 284 kPa to be discharged with an rFIS of 0.

Two patients actually suffered this injury twice each, one with a four-year interval between episodes and the other with a five-year interval (Divers 4, 17 and 9, 16 respectively). In both cases, the divers related that, prior to their latest injury, they had had some degree of residual hearing loss from their initial accident which had worsened acutely when they presented with their second episode of IEDCS. Only one patient in this study population had been evaluated for the possibility of having a PFO and that finding was positive.

## Discussion

The history of the initial observations and the evolution of the establishment of IEDCS as a discrete clinical entity were very nicely summarised by Edmonds in a previous edition of this journal.[19] Prior to the early 1990s, this malady was thought to be mostly seen in those who undertook deep, mixed-gas diving and was rarely seen in compressed air divers. Doolette and Mitchell recently described the possible biophysical basis for the development of inner ear DCS in deep, mixed-gas divers.[20] In the past two decades, however, reports have emerged suggesting that isolated IEDCI may also be seen in recreational divers using compressed air at shallower depths than had been noted previously.[9,10] In a later series of 29 sport divers using compressed air, 28 evinced isolated inner ear DCS.[11]

Thus, it is reasonable to conclude that while isolated IEDCI was predominantly seen in deep, mixed-gas divers, it was not exclusively confined to those divers and may have been more common than previously thought and simply not reported. Indeed, this case series includes 12 cases who were treated in the 1980s. It could also have been that actual cases of IEDCI occurring in air-breathing divers were dismissed as improbable and assigned to another diagnosis because the then published literature implied that it could not happen in air divers or, if it did, only in very rare circumstances, even though some of the earliest accounts of this problem had actually been observed in air divers.[1,5,6] Since that time, there has been a substantial increase in the numbers of recreational divers, most of whom continue to use air as their breathing medium, and so it stands to reason that there might be an increase in the numbers and frequency of this entity as a result.

One of the major challenges to unmasking this condition is the ability to clarify the diagnosis of IEDCI and differentiate it from inner ear barotrauma, which can present with similar symptoms.[21] Oftentimes this requires comprehensive audiological and vestibular testing (audiogram, tympanometry, electronystagmography) to more precisely and objectively evaluate and diagnose cases accurately. As was pointed out by Wong and Walker, it can be quite difficult to organise and execute precise and timely neuro-physiological testing when confronted with a patient who is acutely symptomatic and may need to be expeditiously recompressed.[8] This places the burden of making that initial assessment on the diving medical physician, who needs to ferret out the diagnosis based upon a careful and detailed history of the incident event(s) and a thorough and accurate physical exam.[22]

This study is hampered to some degree by the fact that only eighteen (64%) cases had additional audiological and neuro-physiological studies and/or evaluation. Such studies would have been particularly useful in diver 14, in whom nystagmus was not discernable, diver 24, with isolated hearing loss, and diver 25, who had neither discernable nystagmus nor hearing loss, as they may have provided additional objective findings upon which to predicate the diagnosis. However, the physical findings alone in most cases supported the diagnosis of an acute peripheral cochleo-vestibulopathy,[16] with IEDCI being the most likely underlying aetiology.

With the more recent reports, yet another perplexing and potentially significant association between the existence of a PFO and IEDCI has been suggested.[12–14] This implies that an isolated inner ear peripheral cochleo-vestibulopathy could result from direct embolisation of the anterior vestibular

artery or its branches. This theory, if true, would support the mechanism of injury in cases 15, 25 and 28, suspected of having arterial gas emboli resulting in IEDCI. Since those initial PFO reports, only one case in which the diving history did not seem to support the development of IEDCI has been seen at the HTC. That patient was sent subsequently for contrast echocardiogram to evaluate the existence of a PFO, and the results were positive. In retrospect, it is possible that some of the divers with IEDCI following less than provocative dive profiles may also have had a PFO which may have contributed to their specific episodes. That question will necessarily go unanswered.

This case series reaffirms most of the findings of previous studies. The presenting symptoms and physical findings were not uniform.[4] The most common symptom was vertigo, followed by nausea and vomiting. With respect to physical findings, nystagmus and postural imbalance were the most common findings. Hearing loss was seen in only about one third of the cases, while both tinnitus and hearing loss were observed in just two cases.

The average depth of the incident dive might be considered to be shallow in comparison with previous reports.[9,10] However, the majority of cases emanated from multiple deep dives without surface intervals, thereby resulting in a considerable accumulation of inert gas, or from seriously violated dive tables with inadequate decompression. The average time to onset of symptoms in this study group was similar to that reported by Nachum et al.[11] Symptoms developed after the incident dive as opposed to during it, and none had experienced an event or had physical findings suggestive of otic barotraumas. This is a presentation more consistent with IEDCI than inner ear barotrauma.[22]

A rather stark relationship between delay to treatment and eventual outcome has been reported, with a delay in excess of 42 minutes resulting in residual inner ear dysfunction in that series.[6] This may explain those cases with residual impairment in this study but, conversely, the proportion of cases who obtained clinically observable recovery was slightly better than that of the findings of Nachum.[11] This suggests that the window of opportunity to effect meaningful treatment may be considerably longer than originally thought. Delay to treatment was most often a consequence of the logistics involved in transporting cases to Oahu from the neighboring islands.

Another recommendation in the study by Farmer was the use of deeper treatment schedules to treat IEDCS.[6] In all but three of the cases in this study, the deep treatment schedules routinely employed at the HTC were used. The use of the HTC deep tables in the majority of cases may explain the higher rates of clinical recovery in this study compared with the other series in which shallower treatment tables were employed.[6,11,23]

In this case series, there were no patients who failed to show some clinical improvement. Insofar as there is natural compensation for such injuries mediated via the cerebellum that occurs over time, it may be difficult to ascribe clinical recovery to treatment alone. Typically, it is believed that most vestibular end-organ injuries result in permanent damage and that full compensation may take two to four weeks to occur.[24] That most cases clinically improved within a week's time of recompression therapy may indicate only that it accelerated the normal compensatory mechanisms. However, given the significance of this injury, it is doubtful that any diving medical officer would rely solely on spontaneous recovery and not treat these cases with recompression. Recovery from a hearing deficit is best followed by serial audiological testing.

Finally, in the present series, the role of PFO cannot be elucidated since it was investigated in only one diver. Three cases were thought to result from direct embolisation.

## Conclusions

IEDCI can and does occur in recreational scuba divers using compressed air. It accounted for nearly 3% of all DCI cases treated at the HTC, which was a lower frequency than that reported by others.[25] While IEDCI is more likely to occur after more extreme exposures to depth and dive times, it may occur from shallower exposures and from embolisation. Significant impairment with a high potential for permanent incapacitation exists for those cases not treated early and aggressively to include complete initial and follow-up oto-neurological evaluation and testing. Our view is that, in line with Farmer et al,[6] deeper treatment schedules should be employed to treat IEDCI whenever possible. In the light of recent evidence, divers with IEDCI should probably undergo contrast echocardiography to exclude PFO, especially those for whom there is not a more compelling explanation to account for their symptoms.[12–14]

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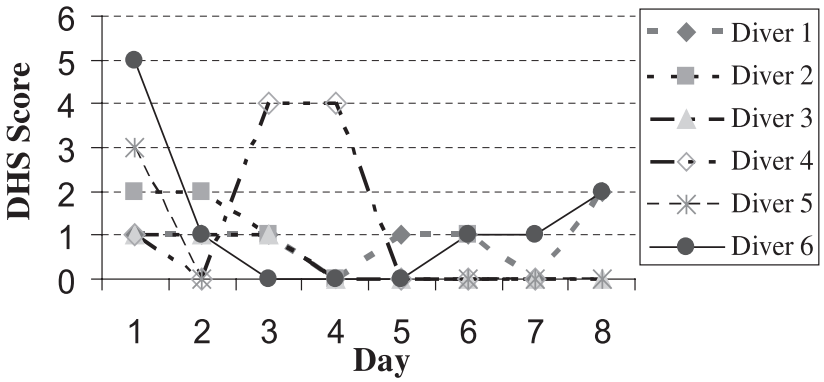
**Figure 2**
**Diver health scores (DHS – Diver Health Status)**




**Erratum**

Fock A. Health status and diving practices of a technical diving expedition. *Diving and Hyperbaric Medicine*. 2006; 36: 179-85.

Figure 2 did not reproduce correctly, and is republished here.