# Vestibular rehabilitation and recovery in divers with inner ear decompression sickness: a case series

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**Keywords**
ENT; Fitness to dive; Scuba diving; Sharpened Romberg test; Vertigo

**Abstract**
(Stokes RJ, Watts D, Smerdon G, Hall SD, Bunn L, Marsden J. Vestibular rehabilitation and recovery in divers with inner ear decompression sickness: a case series. Diving and Hyperbaric Medicine. 2025 30 September;55(3):236–245. doi: 10.28920/dhm55.3.236-245. PMID: 40986919.)
**Introduction:** The mechanism of injury and recovery of divers with inner ear decompression sickness (IEDCS) is not well understood and there is no consensus regarding management following recompression treatment. Given the rare occurrence, divers are not routinely offered the standard therapies that patients with other acute vestibular disorders may be offered such as vestibular rehabilitation.
**Methods:** This is an observational case series of 13 divers presenting acutely with IEDCS to DDRC Healthcare in Plymouth, UK between July 2021 and January 2024. Vestibular and balance tests were undertaken to aid the treating dive physician in the diagnosis and management of the divers with both hyperbaric oxygen therapy and customised vestibular rehabilitation.
**Results:** Average values for vertical perception, posturography, dynamic gait index and patient-reported outcomes measures improved by discharge and at the three month follow up despite 67% showing an ongoing positive head impulse test or nystagmus in the dark on videonystagmography at follow up.
**Conclusions:** Divers should be warned that despite symptom resolution or minimal residual symptoms post-IEDCS there is a high rate of deficit evident on vestibular testing, and this, alongside investigation for a right to left cardiac shunt, should be a major consideration when considering returning to diving. For the clinician, a stopwatch timed Sharpened Romberg's test appears to be a reasonable method for monitoring progress of balance stabilisation during the treatment period. Early initiation of vestibular rehabilitation exercises should be considered for all divers with IEDCS.

## Introduction

Inner ear decompression sickness (IEDCS) represents around 20% of decompression sickness cases.[1] This may present with acute vestibular symptoms (vertigo, unsteadiness) and/or cochlear symptoms (loss of hearing, tinnitus), ranging in severity from a feeling of unsteadiness to severe vertigo and vomiting. These symptoms can be resistant to recompression therapy, requiring repeated treatments and many divers are left with permanent deficits in vestibular function.[2] The exact mechanism of injury and recovery is not well understood. Animal studies have shown that there is haemorrhage and precipitation of blood proteins into the perilymphatic and endolymphatic vestibular systems, as well as ectopic new bone growth in fluid spaces after one month.[3,4]

Diver case studies have described IEDCS after deep/technical diving where tissues are supersaturated with inert gas. In some cases, the event was associated with the switching of breathing gases causing local counter-diffusion of inert gases and exaggerated supersaturation which the inner ear appears particularly sensitive to.[5] Other studies describe conservative dive profiles well within table limits. In such cases, around 70% of divers are found to have an underlying right to left cardiac shunt such as a patent foramen ovale (PFO) suggesting a possible embolic pathology.[6,7]

IEDCS is treated acutely with hyperbaric oxygen recompression therapy with better outcomes seen with a shorter time to recompression.[8] Therapy is repeated daily until the symptoms improve or plateau. Diving physicians commonly use basic clinical bedside balance tests such as a heel-toe walk or a Romberg/Sharpened Romberg test to assess symptoms, however there is no standardised method for comprehensively assessing residual vestibular symptoms.[9] More detailed vestibular testing can be undertaken but this is often limited by access to specialised clinicians and equipment.

Previous studies have utilised detailed vestibular testing on divers presenting acutely with inner ear pathology. Results have shown the characteristics of a peripheral vestibular lesion but these studies have not provided a comprehensive insight into the specifics of the mechanism of injury or how the lesion/symptoms change over time in these divers.[2,10] Studies regarding IEDCS tend to focus on the acute phase, with little understanding of how function is affected over the longer term and what considerations should be made when assessing fitness to return to diving. There is also a paucity of qualitative information regarding the burden of residual symptoms.

Vestibular rehabilitation exercises are commonly used in other acute vestibular disorders such as vestibular hypofunction, but they have not been utilised in IEDCS recovery as standard.[11] Early initiation of vestibular rehabilitation has been shown to aid clinical recovery in these disorders and should be considered an important adjunct to hyperbaric therapy.[12]

The aim of this case series was to describe the short and long-term effects of IEDCS on the vestibular system in these divers to aid understanding of symptom burden, prognosis and allow a more informed assessment when considering fitness to return to diving.

## Methods

Ethical approval for studying the divers with IEDCS was obtained through the Integrated Research Application System (Approval number 337421). Ethical approval for collection of normative data was obtained through Plymouth University. An observational study was performed involving all scuba divers presenting acutely with IEDCS to the DDRC Healthcare hyperbaric chamber in Plymouth between July 2021 and September 2024. Any alternative diagnosis for inner ear symptoms such as barotrauma or infection was an exclusion criterion and thorough history and otoscopic examination was undertaken on each diver presenting at the hyperbaric centre.

Divers were offered laboratory and clinical vestibular testing (as described below) as well as a personalised vestibular rehabilitation exercise program to aid their diagnosis and recovery. Laboratory based vestibular testing was offered after the initial recompression and once symptoms (e.g., vomiting/severe nausea) allowed. Testing was timed to not interfere with scheduled repeated recompression treatments, with results used to aid clinical assessment by the treating dive doctor. Testing was repeated on the day of discharge with advice to progress rehabilitation exercises at home and the option of telephone follow up. Testing was also repeated opportunistically when divers attended DDRC Healthcare for their return to diving medical examination which was usually scheduled for three months post injury.

Assessments included clinical tests (head impulse test, eye position with and without Frenzel's glasses and eye movement [smooth pursuit and saccades], timed Romberg's in tandem stance and the dynamic gait index [DGI]). The DGI assesses eight walking tasks on a 0–3 ordinal scale including head movements while walking. The maximum score is 24 with greater than 22 indicating safe ambulators and < 19 being predictive of falls.[13]

Laboratory vestibular tests included videonystagmography (VNG) and rotary chair assessment of nystagmus, eye movements (smooth pursuit, saccades and optokinetic reflex) and vestibulo-ocular reflex (VOR) (using sinusoidal and step rotations) as described previously.[14]

In addition, posturography measured postural sway over 30 seconds via force plates with a 1 kHz sampling rate (Moteklink, Motek, Netherlands or Kistler 9286AA Kistler, Instruments Ltd, Hampshire, UK) with eyes open/closed with feet 10 cm apart, feet together or in tandem. Signals were filtered (4th order, 5 Hz low pass Butterworth) and the centre of pressure velocity ($mm \cdot s^{-1}$) was calculated offline.[15] Subjective visual vertical was assessed in the dark with a static background and a dot array rotating at $30° \cdot s^{-1}$ clockwise or anti-clockwise using the Rod-and-Disc test.[16] Divers also completed a set of questionnaires validated for people with peripheral vestibular dysfunction comprising the Dizziness Handicap Inventory,[17] Vertigo Symptom Scale,[18] Situational Characteristics Questionnaire,[19] and the Activities-Specific Balance Confidence Scale (ABC-UK).[20] Open discussion regarding any issues highlighted on these questionnaires allowed for qualitative feedback of the divers' experience of their symptom burden and progress with vestibular rehabilitation.

Normative data for the posturography, Rod and Disc test and VNG/rotary chair testing was collected using members of the general population as controls ($n$ = 15). Those who had a history of decompression illness or vestibular disorders were excluded. The IEDCS data was defined as abnormal if it was greater or lesser than the mean ± one standard deviation (SD) of the normative values.

## Results

### DIVER DEMOGRAPHICS AND PROVOKING FACTORS

Between July 2021 and September 2024 thirteen divers were seen with symptoms of inner ear disorder after diving. One diver was excluded due to an alternative diagnosis of inner ear barotrauma; this diagnosis was made after worsening of symptoms at pressure. Mean age of the 12 divers was 53 years (range 32–73), 58% were male and 42% female. Mean depth of the provoking dive was 27 metres of seawater (range 19–36); 10 divers were breathing air and two were breathing nitrox 32%. All dives were uneventful with no rapid ascents or known missed decompression

stops. In terms of contributing risk factors; six divers had been diving on consecutive days, two were overweight, one had dehydration secondary to gastroenteritis, one was fatigued from strong currents during the dive, one had type 2 diabetes mellitus and one had a history of previous IEDCS. Of the nine divers who went on to have a bubble contrast echocardiogram, six had an underlying PFO (67%).

## SYMPTOM ONSET AND MANAGEMENT

Time from the end of the dive to the onset of symptoms showed considerable variation (mean four hours, range 0–19). The mean time from symptom onset to first recompression treatment was nine hours (range 5–15) with two outliers excluded (76 and 312 hours). These outliers were divers who did not recognise that their symptoms were decompression sickness and delayed seeking medical attention. Two divers had a delay of over 10 hours before recompression; one required imaging in the emergency department prior to reaching the chamber and the other had put his symptoms down to seasickness whilst he had been on the dive boat. All of the divers were treated with an initial Royal Navy Treatment Table 62 at 284 kPa (2.8 atmospheres absolute). Mean number of hyperbaric treatments was seven (range 1–12). All divers were given a balance retraining booklet[21] on the first day of their presentation and then more personalised exercises were given following vestibular and balance assessments.

## VESTIBULAR TESTING

Of the 12 divers with IEDCS, eight were seen at all three test points; one was excluded as they were not seen in the acute phase, one missed testing due to equipment malfunction and two did not attend follow-up. Initial testing (referred to as VFT1) was performed at a mean of 2.13 days post symptom onset (SD 1.13), discharge testing (VFT2) was performed at 9.63 days (SD 4.81) and follow-up testing (3MFU) at 6.53 months (SD 5.19).

Although no baseline audiometry results were available for comparison, no objective signs of new hearing loss were seen on audiogram for any of the eight divers. All had signs of vestibular dysfunction; 63% had a right sided lesion whilst 38% had a left sided lesion (as determined by the head impulse test).

**Table 1**

Results of dynamic gait index and vestibular function testing at admission, discharge and three-month follow-up; VOR – vestibulo-ocular reflex

| Measure | Admission ($n$ = 8) | Discharge ($n$ = 8) | Follow-up ($n$ = 8) |
|---|---|---|---|
| Mean time of assessment after onset | 2.13 days | 9.63 days | 6.53 months |
| Dynamic gait index, mean (range) | 19 (16–23) | 23 (20–24) | 24 (23–24) |
| Optokinetic response | 10% asymmetry towards side of lesion | 3% asymmetry towards side of lesion | Symmetrical |
| Smooth pursuit gain 0.2 Hz–0.4 Hz | Normal | Normal | Normal |
| **Nystagmus slow phase velocity** | | | |
| Centre | Abnormal | Normal | Normal |
| Away from lesion | Abnormal | Normal | Normal |
| Towards lesion | Normal | Normal | Normal |
| **Saccadic** | | | |
| Accuracy away/towards lesion | Normal | Normal | Normal |
| Latency away/towards lesion | Normal | Normal | Normal |
| **VOR sinusoidal rotation** | | | |
| Gain 0.2 Hz ($n$ = 8) | Normal | Normal | Normal |
| Asymmetry 0.2 Hz ($n$ = 8) | Abnormal | Abnormal | Abnormal |
| Gain 0.32 Hz ($n$ = 7) | Normal | Normal | Normal |
| Asymmetry 0.32 Hz ($n$ = 7) | Abnormal | Normal | Abnormal |
| **VOR step rotation** | | | |
| Time constant away lesion (per) | Normal | Normal | Normal |
| Time constant towards lesion (per) | Normal | Normal | Normal |

*Videonystagmography (VNG)*

Clinical outcomes are summarised in Table 1 with a supplementary version providing the mean (SD) and normative data.* Nystagmus was present in all divers in the dark at initial presentation along with a positive horizontal head impulse test to the side of the lesion. Nystagmus in the dark (Table 1 and Figure 1A) showed higher slow phase velocity with gaze towards the contralesional side compared to the ipsilesional side, as expected with Alexander's law.[22] Nystagmus towards both sides decreased in velocity by the end of the treatment period (Figure 1A).

At initial presentation the optokinetic response showed a higher gain when the stimulus moved toward the ipsilesional side but there was a large standard deviation. By discharge and follow up the response was more symmetrical (Table 1). Smooth pursuit gain and phase along with saccadic latency and amplitude were within normal limits at initial and subsequent testing (Table 1).

The VOR was tested using sinusoidal harmonic acceleration in a rotary chair. With rotation at both 0.2 Hz and 0.32 Hz in the dark, gain was normal but low when compared to control participants and remained low over all testing points (Table 1 and Figure 1B), however there was large individual variation. Asymmetry improved over time (Table 1 and Figure 1C). Step rotation (140°·s$^{-1}$ acceleration/deceleration with a 60°·s$^{-1}$ fixed-chair velocity) showed a shorter time constant when rotating toward the ipsilesional side, reflecting an impaired VOR. Although not statistically significant, the time constant towards the ipsilateral side appeared to increase over the treatment period whereas the time constant on the contralateral side appeared to decrease, resulting in a more symmetrical VOR (Figure 1D).

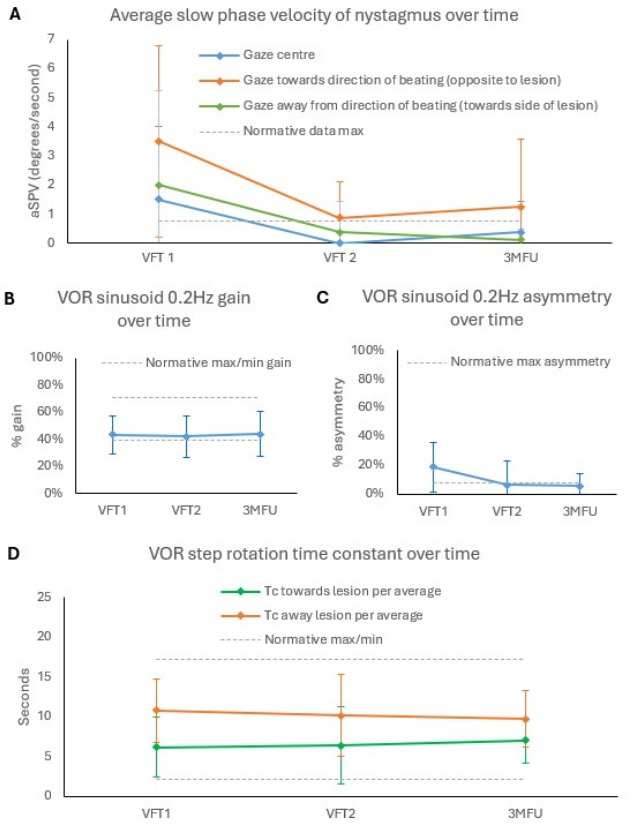
Five of the eight divers (62.5%) showed a persistent deficit on follow up testing with a positive head impulse test or presence of nystagmus with gaze in the dark.

*Balance and walking*

The velocity of sway (Posturography testing) with a 10 cm stance width (eyes open or closed) or with feet together and eyes open showed little variation over time. In contrast 'feet together, eyes closed' and 'tandem, eyes open' were the most sensitive tests, being increased at baseline and showing improvements over time (Figure 2A and B). 'Tandem, eyes closed' was very difficult for the divers to maintain and only two were able to hold the stance at all at initial presentation, and three at discharge and follow up. Those that did manage to hold tandem stance eyes closed showed improvement over the treatment period in both time in stance and measured sway (Figure 2A and B).

**Figure 1**

Videonystagmography results (mean and standard deviation) showing evidence of a peripheral nerve lesion; 3MFU – three-month follow-up; SPV – slow phase velocity; Tc – time constant; VFT1 – vestibular function testing at admission; VFT2 – vestibular function testing at discharge; VOR – vestibulo-ocular reflex




The DGI score improved over time with a mean score of 19/24 (IQR 4) at initial presentation, 23/24 (IQR 2) at discharge, and 24/24 (IQR 0) at follow up (Table 1 and Figure 2C).

*Subjective visual vertical*

Subjective visual vertical using the Rod & Disc test[16] showed little deviation with a static background. However, with a rotating background there was an asymmetric response with greater deviation when the background rotated towards the ipsilesional side. This improved over time (Figure 2D).

*Patient reported outcomes (PROMS) and subjective report*

Scores for all questionnaires improved over time with the minimal detectable change reached for the dizziness handicap inventory (> 18 points)[23] and the ABC-UK Scale (> 15%)[24] (See Table 2 and Figures 3A–D). At long term

***Footnote:** Supplementary version is available to download from https://www.dhmjournal.com/index.php/journals?id=361

**Figure 2**

Posturography (A+B) (note the difference in scale between A and B); dynamic gait index (C), and subjective visual vertical (D); data are shown as means and standard deviation. 3MFU – three month follow-up; FAEC – feet apart eyes closed; FAEO – feet apart eyes open; FTEC – feet together eyes closed; FTEO – feet together eyes open; TEC – tandem eyes closed; TEO – tandem eyes open; VFT1 – vestibular function testing at admission; VFT2 – vestibular function testing at discharge

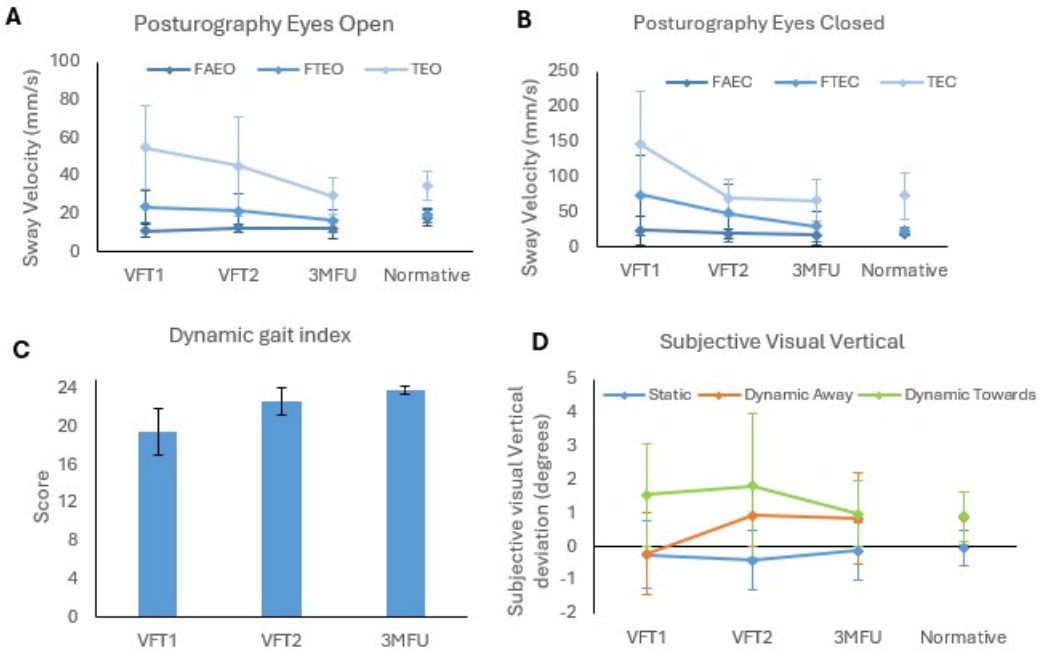


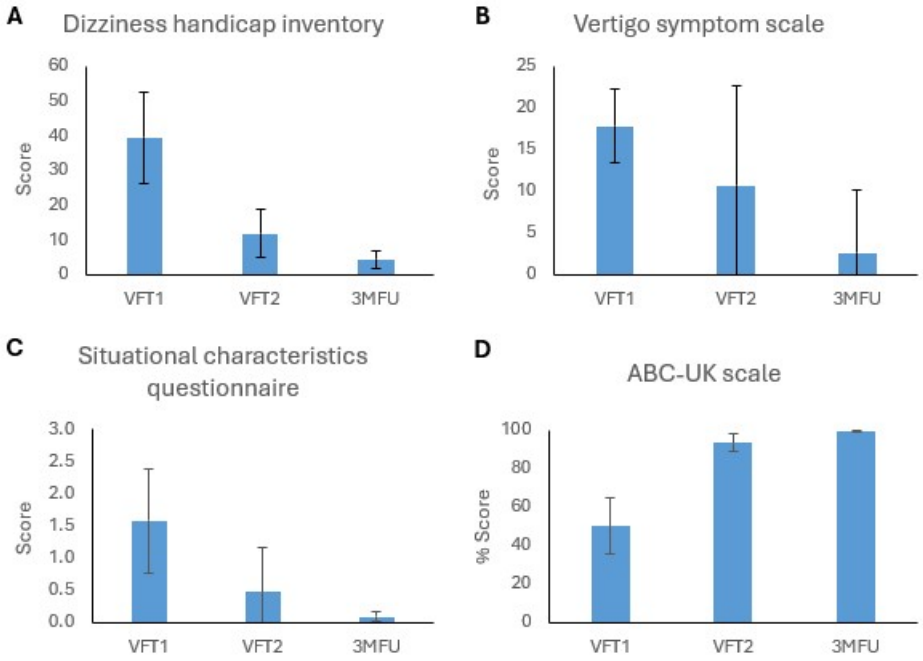

**Table 2**

Mean values for the patient reported outcome measures at admission, discharge and three-month follow-up

| Measure | Admission ($n$ = 8) | Discharge ($n$ = 8) | Follow-up ($n$ = 8) |
|---|---|---|---|
| Dizziness handicap inventory[17]<br>> 10 = refer to balance specialist<br>16–34 = mild handicap<br>36–52 = moderate handicap<br>≥ 54 = severe handicap | 39.50 | 11.75 | 4.29 |
| Vertigo symptom scale<br>Maximum = 60<br>> 12 = severe dizziness[36] | 18.25 | 7.88 | 1.71 |
| Situational Characteristic Questionnaire | 1.58 | 0.47 | 0.08 |
| Activities-specific balance confidence scale[37]<br>Level of physical functioning, total = 100%<br>80% = high<br>50–80% = moderate<br>< 50% = low | 50.02% | 93.46% | 99.38% |

**Figure 3**

Patient reported outcome measures (A-D) showing clinical improvement over time; data are shown as means and standard deviation. 3MFU – three-month follow-up; ABC-UK scale – activities-specific balance confidence scale; VFT1 – vestibular function testing at admission; VFT2 – vestibular function testing at discharge




follow up the divers anecdotally reported the return of some symptoms with fatigue, alcohol consumption, viral illness, and low light environments.

*Personalised vestibular rehabilitation*

All divers reported finding the rehabilitation exercises useful both in the acute phase and once discharged home. Customised exercises tended to focus on gaze stability, visual desensitisation and balance exercises in standing and walking in conditions of varying sensory information.[25,26] Complexity was increased by (a) increasing the speed of movement, (b) reducing the base of support and postural starting position, and (c) reducing the available sensory cues (e.g., eyes closed, removal of light touch support and standing on foam). Exercises targeted specific functional difficulties (e.g., standing at heights and painting) and were integrated in function task simulations/ activities. Exercises were prescribed for 5–10 minutes twice a day.[26] Visual dependence was determined through the Situational Characteristics Questionnaire, large (> 2 degree) visual vertical deviations with a moving visual scene and clinically, through a significant symptom increase when undertaking exercises when a moving visual scene was added. Visual dependence was addressed through visual desensitisation where exercises were performed in front of visual motion scenes of increasing complexity.[27]

*Return to diving*

Of interest, of the eight divers we have the information for, six returned to diving whilst two (with confirmed PFOs) were advised not to return to diving based on their residual subjective symptoms (including the diver who had a previous episode of IEDCS). Of those who returned to diving, four had confirmed PFOs; two had closures whilst the other two opted not have closures and returned to diving using recommended conservative strategies.[28] The two divers with negative bubble echocardiogram tests returned to diving with added conservatism. The six divers who returned to diving had no residual symptoms or signs of vestibular dysfunction on the standard dive medical examination. To date, we have not had reports of any further episodes of decompression illness.

## Discussion

The mean age of the divers (53 years old) was not dissimilar to the average age of UK divers reported in diver surveys (~47 years old).[29] The gender of the divers was slightly more balanced than expected as there tends to be a larger male cohort in UK diving.[29] The average depth of the provocative dives was in line with previous case series of IEDCS[30] and the prevalence of an underlying PFO aligned with the average of 70% seen in other series.[31,32] The delay

to recompression (nine hours) was similar to cases in Israel (8.54 hours with those ≥ 72 hours excluded as outliers).[2]

At presentation all divers had evidence of a purely peripheral dysfunction with no central deficits seen on clinical or VNG testing. This was the case for divers presenting with purely vestibulo-cochlear symptoms as well as those with other symptoms of decompression sickness. Of the two divers with other symptoms, one had neurological signs (left leg weakness that rapidly resolved with oxygen administration), and one had cutaneous decompression sickness.

VOR gain following sinusoidal rotation increased by 7–8% over time. As rotary chair testing is expensive this test is not be used routinely in divers. Further, as rotation occurs at low velocities it is assessing both lateral canals and is therefore not as useful for diagnosing unilateral vestibular lesions compared to bilateral lesions, which are vanishingly rare in IEDCS. However, the step rotation test does allow an assessment of the time constant that involves both peripheral and central components and thus provides a possible window into central compensation processes. These showed a decrease when rotating to the side of the lesion and an increase when rotating away from the lesion over time suggestive of central compensatory changes.

Testing of visual perception with a static background showed little deviation. A deviation here may indicate deficits in utricle function.[33,34] Dynamic testing (with a moving background) did show changes with a greater visual vertical deviation when the background rotated towards the ipsilesional side. This reflects movement of the eyes in the direction of the VOR that drives the accompanying nystagmus. Whilst the dynamic results were as expected, the deviation was within the 2° error quoted in the literature and therefore should be interpreted with caution.[33,34]

Optokinetic nystagmus gain immediately after the lesion was higher when the stimulus moved towards the side of the lesion reflecting the asymmetry in the VOR response. However, as the optokinetic response requires a stimulus that covers the whole visual field, and the gain can be open to factors such as attention and instruction it is clinically less reliable.

Symptomatic improvement, as evidenced by the patient reported outcomes and posturography results, was seen in all divers despite an ongoing peripheral deficit in vestibular function seen in the head impulse test ± videonystagmography in five of the eight divers. This level of residual dysfunction (~70%) has been documented in other studies with divers with IEDCS[2,30] and should be a major consideration when assessing fitness to return to diving especially as the underwater environment can potentially contribute to disorientation. Poor visibility, swell, reduced visual reference and the potential for further insult to the inner ear (barotrauma, alternobaric vertigo, further IEDCS) could result in a hazardous situation.

Vestibular compensation is a well-documented phenomenon after a peripheral vestibular lesion with mechanisms involving adaptation, sensory substitution (central) and habituation identified.[27] VNG testing with step rotation showed a more symmetrical slow phase velocity time constant when comparing the ipsilateral and contralateral rotation over time. We hypothesise that over time central compensation focused on the brainstem structures involved in velocity storage (cerebellum, medial vestibular nuclei and prepositus hypoglossi nuclei) resulting in a shortened time constant on the contralateral side and reductions in left-right asymmetries that mediate occulomotor, perceptual and balance symptoms. We further hypothesise that targeted rehabilitation exercises could facilitate compensatory mechanisms to promote faster functional recovery.

Vestibular rehabilitation exercises should be considered for all divers presenting with IEDCS as a useful tool to aid recovery and in case of recurrence of symptoms after discharge. They are utilised in other unilateral vestibular diseases such as post neurectomy/schwannoma removal and vestibular neuritis with the mantra of 'the earlier the rehabilitation, the faster the recovery'. This reflects the presence of a critical period immediately after a lesion in animals where early engagement of the vestibular system leads to better recovery than delayed intervention (e.g., by 1–2 weeks).[27,35] In terms of the practicality of this, in the acute presentation of IEDCS we found that divers could be given the balance retraining booklet[21] to read during their first hyperbaric treatment in the chamber and to take away with them. This meant that they started the exercises on day one and were engaging in the rehabilitation process straight away. Once seen for the vestibular testing, they were given more personalised exercises including balance exercises and gaze stabilisation.

Dependence on vision was identified either clinically, via the Situational Characteristics Questionnaire or the vertical perception test. Very visually dependent people have a poorer spontaneous vestibular compensation as they tend to over rely on visual information for balance and orientation rather than using multi-sensory information (i.e., vision, somatosensory and remaining vestibular) and have difficulty resolving conflicts between vision and vestibulo-proprioceptive information. When using vision in isolation, errors can arise in interpreting self-motion from motion of the environment.[25,27,35] If visual dependence was identified, the strategy for rehabilitation included use of optokinetic videos and stimulus alongside the other exercises. Optokinetic training and stimulus videos are freely available online. We chose videos involving walking down a supermarket aisle or along a crowded street. These were displayed on a large

screen and the diver was initially asked to stand watching the video and describe their symptoms. If they were able to do this with ease, then the exercise progressed in difficulty with the diver asked to walk towards the screen whilst keeping fixated on the moving stimulus. This then progressed again by walking towards the screen and moving the head from side to side. This exercise was easy to recreate in the home environment and the divers could pick videos they found particularly provoked their symptoms.

Posturography demonstrated that use of the sharpened Romberg's (tandem eyes closed) was a reasonable method of assessing balance as a bedside test. Timing the duration of holding the stance using a stopwatch is recommended as a measurement of improvement (with consistency of self-selected leading foot and number of trials allowed).

The mean dynamic gait index (DGI) score at the initial testing point was 19 out of 24 which was better than expected as at initial presentation these divers often require a lot of support with standing and walking due to the vertigo and nausea (Table 1 and Figure 2A). However, it is plausible that this could be indicative of some improvement over the first day following initial hyperbaric recompression treatment as the testing session typically took place following this (to not delay treatment). As a diving physician, performing the DGI was a useful method of identifying movements that provoked symptoms and therefore formed a crude identification of initial exercises to suggest for rehabilitation. All divers scored 24 at follow-up which may represent a ceiling effect and a limitation in using the DGI. Going forward, the DGI pre- and post- the initial hyperbaric treatment will be checked to establish any improvement over this time. Over the three testing periods the overall change in DGI scores was five points, exceeding the minimal detectable change of four points quoted in the literature.[13]

The patient reported outcome measures were useful for identifying specific activities that the divers struggled with however, as they were designed for more chronic vestibular disorders, many of the questions were not applicable. A questionnaire designed for divers that covers symptoms occurring in both acute and chronic stages would provide better representation of this patient cohort.

This observational study of a case series documents real world practice therefore the number and timings of assessments was determined largely to target information for the clinical team rather than part of an investigative study. Choice of vestibular testing was based on available equipment rather than by design allowing only for investigation of the horizontal semicircular canals. Use of video head impulse testing and vestibular evoked myogenic potentials (VEMPS) will allow for assessment of the vertical semicircular canals and the otolith organs. Further research utilising these techniques is planned along with qualitative data collection.

## Conclusions

Early initiation of vestibular rehabilitation exercises should be considered for all divers with IEDCS. Divers should be warned that there is a high rate of residual signs of vestibular deficit and that factors such as viral illness or alcohol consumption may interfere with the central compensation process even years after their injury. This deficit should also be a major consideration when considering returning to diving due to the risk of further decompression illness or barotrauma to the inner ear and poor environmental conditions inhibiting compensatory mechanisms. For the clinician, a stopwatch timed Sharpened Romberg's test (best of three with consistency of preferred leading leg) appears to be a reasonable method for monitoring progress of balance stabilisation during the treatment period. Clinical assessment should involve head impulse test and assessment of nystagmus with/without Frenzel's glasses. All divers should be educated regarding the possibility of an underling right to left cardiac shunt and offered referral to a cardiac specialist. Further investigation is needed to understand more about the mechanism of injury and recovery in IEDCS.

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**Conflicts of interest and funding**

This work was funded by the University of Plymouth and DDRC Healthcare as part of a PhD research programme. No conflicts of interest were declared.

**Submitted:** 18 March 2025
**Accepted after revision:** 22 June 2025