# ORIGINAL PAPERS

## DECOMPRESSION ILLNESS IN THE TUNA FARM DIVERS OF SOUTH AUSTRALIA

Pauline Whyte

**Key Words**

Decompression illness, hyperbaric oxygen, occupational diving, risk, safety, treatment, treatment sequelae.

**Abstract**

The Hyperbaric Medicine Unit of the Royal Adelaide Hospital treated a total of 22 tuna farm divers with decompression illness (DCI) between 1992 and 1998. Overall two thirds of the divers were left with sequelae after treatment. The diving practices of the tuna farming industry, the likely reasons for DCI, the treatments used and the results obtained are discussed. In 1997 regulations were introduced to raise the standards of training and dive management in the industry. Since then the incidence of DCI has dropped but the clinical presentations are unchanged. The Royal New Zealand Navy scoring system for DCI severity and treatment response was used to describe the clinical course of these patients.

### Introduction

In 1992, in response to a 67% reduction in the tuna catch quota in South Australia, fishermen in the Port Lincoln area began farming tuna and by 1998 fourteen tuna farming operations had been developed. From December to February tuna are caught in the Great Australian Bight and towed in cages, at approximately 1 knot, to Port Lincoln where they are transferred to stationary pens. The tuna are fed pilchards for 1-8 months before being hand harvested for the Japanese market where a 40 kg fish can earn up to $1,000.

Approximately 40 divers are employed full time, increasing to 60 during the catching season. They inspect, maintain and repair cages, remove dead fish and occasionally sharks from the enclosures and monitor the herding, feeding and hand harvesting of the tuna.

Farming procedures were developed by fishermen with little knowledge of diving. Divers usually had only recreational training, if any. Diving was conducted with little consideration of the risks of decompression illness (DCI). Divers relied almost solely on surface air supply from petrol driven compressors with no back-up systems, full-face masks or voice communications. Multiple ascents were typical, with divers coming to the surface repeatedly to receive or to give instructions.

By early 1995, WorkCover Corporation (South Australia's Workers Compensation Authority) had received 39 diving related claims, $1,475,326.00 in compensation had been paid and 17 divers had been treated for DCI at the Royal Adelaide Hospital (RAH).

In response, the Department of Industrial Affairs and WorkCover Corporation implemented strategies to raise the standard of diving. Inspections of the tuna farms and their diving practices were carried out during 1995. Training sessions were conducted for divers, supervisors and employers and safer diving procedures established. However, the death of an untrained scuba diver in March 1996 highlighted the need for further intervention and in March 1997 the Government introduced the Approved Code of Practice for Tuna Farm Diving based on AS2299 (Australian/NZ Standard 2299 for Occupational Diving). All divers are now required to be occupationally trained and the South Australian Underwater Training School, established in Port Lincoln in 1996, now operates under a Memorandum of Understanding with the Australian Fisheries Academy. The widespread introduction of full-face masks allows continual communication with the diving supervisor and surface crew, thereby reducing the need for multiple ascents. Surface supply gas now has a back up system and divers carry bailout bottles. These interventions have improved the standard and safety of diving and reduced the number of tuna farm divers presenting with DCI to the RAH.

This report details the nature and severity of decompression illness in the tuna farm divers and the long-term outcome of those affected.

### Data collection

Approval for case note review was obtained from the Ethics Committee and Medical Staff Society, Royal Adelaide Hospital (RAH), and the review was conducted in accordance with the National Health and Medical Research Council Statement on Human Experimentation and Supplementary Notes-1992.

For each diver, age, date of presentation, delay to recompression and whether the diver continued to dive when unwell, signs and symptoms before each hyperbaric treatment, on discharge, at the six weeks follow-up visit and at yearly intervals thereafter, the number and types of recompression therapy received, adjuvant use of lignocaine, results of neuropsychological testing, and fitness to return to diving were noted.

Signs and symptoms were collated and divided into those at presentation, at any time and at the first and most recent follow-up visits. Signs and symptoms present at any time were compared with those of recreational and military divers.[1,2]

One diver presented on two separate occasions and as he made a complete recovery following both episodes of decompression illness, he is included twice in the data. Medical records could not be retrieved for one diver who presented in 1993. Therefore, although seventeen divers presented prior to WorkCover's intervention, data is available for sixteen divers only.

The neuropsychologist's findings were summarised as normal, unlikely organic impairment, possible organic impairment and significant impairment. The time to testing and the results of repeat testing were recorded.

Telephone follow-up established if those unfit to return to diving had found alternative work.

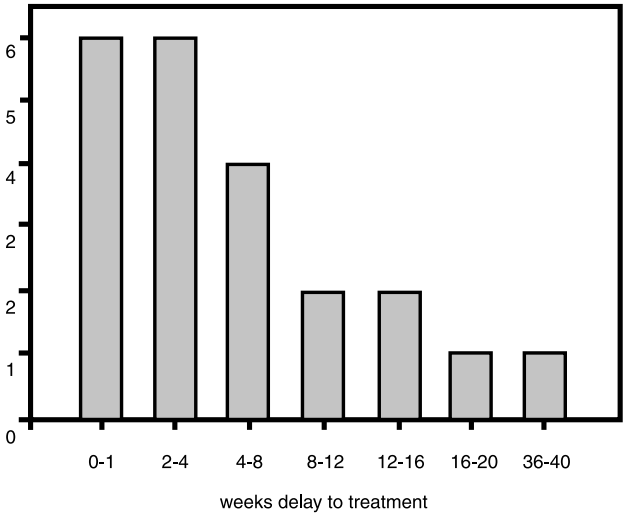


**Figure 1**. Bar chart showing the number of divers in various categories of delay in weeks between becoming unwell and seeking treatment.

**TABLE 1**

**PERCENTAGE INCIDENCE OF PRESENTING SIGNS AND SYMPTOMS IN THREE GROUPS OF DIVERS**

| Signs and Symptoms | Tuna Farm Divers | DAN | Rivera |
|---|---|---|---|
| Pain | 95 | 57 | 92 |
| Paraesthesia | 91 | 52 | 21 |
| Lethargy | 82 | 17 | 1 |
| Cognitive change | 77 | | |
| Objective sensory change | 68 | 52 | |
| Balance | 59 | | 1 |
| Reflexes | 59 | | |
| Headache | 54 | 16 | 4 |
| Mood change | 50 | 3 | 2 |
| Upper limb co-ordination | 36 | | 1 |
| Lower limb weakness | 32 | 22 | 21 |
| Upper limb weakness | 27 | 22 | 21 |
| Dizziness | 18 | 19 | 8 |
| Tinnitus | 14 | 2 | |
| Urinary problems | 14 | 33 | 2 |
| Nausea | 9 | 14 | 8 |
| Lower limb co-ordination | 9 | | 1 |
| Gait | 4 | 10 | |

## Tuna farm divers

Twenty-two male tuna farm divers, average age at presentation 29.4 years (range 19-43 years), were treated for DCI at the RAH between November 1993 and January 1998.

Divers typically had long delays between developing symptoms and seeking medical help (Figure 1). Six divers, who became unwell following a dive and promptly ceased diving, presented within five days. and, for the purposes of this study, were regarded as having "acute" DCI. Two were diving on tuna farms around Port Lincoln, approximately 650 km by road and 250 km by air from Adelaide, and presented to the local hospital. The others developed symptoms while tuna catching and the return to port took between two and four days. All six were retrieved by air, at 1 ATA, to the RAH and received normobaric oxygen and intravenous fluids during transfer.

The remaining 16 divers, referred to the RAH by their local doctors, presented between two weeks and nine months after developing symptoms and, with one exception, continued to dive while unwell. They were regarded as having "chronic" DCI. The term "chronic" does not refer to long-term problems.

## Clinical examination at presentation

At the RAH each diver was assessed. Pain, lethargy, cognitive impairment, paraesthesia and objective sensory change were the commonest manifestations of DCI, occurring in at least 60% of the divers (Table 1). In this study non-specific symptoms, such as lethargy and headache, cerebellar signs, such as balance and co-ordination and manifestations of nervous system damage, such as paraesthesia and objective sensory disorder, all occurred much more commonly than in 1,249 recreational or 935 military divers.[1,2]

The intensity of pain ranged from mild aches to severe pain requiring referral to a Chronic Pain Unit.

**Treatment**

Each diver received a series of recompression treatments until he became symptom free or showed no further improvement with two successive treatments. Three divers received intravenous lignocaine.

The divers received an average of 8 recompression treatments (range 2-14). All divers received a RN 62 as the first treatment table (Table 2) and treatment tables varied thereafter. Treatments 18:60:30, 10:60:30 and 14:90:30 are depth in m: time at depth: ascent time.

**TABLE 2**

**RECOMPRESSION TREATMENT OF TUNA FARM DIVERS**

| Treatment table | Number and % of divers | | Number of treatments |
|---|---|---|---|
| RN 62 | 22 | 100 | 1-3 |
| 18:60:30 | 20 | 91 | 1-10 |
| 10:90:30 | 12 | 54 | 1-8 |
| RN 61 | 7 | 36 | 1-6 |
| 14:90:30 | 3 | 14 | 1-3 |

**Progress**

The incidence of all signs and symptoms had decreased by the first follow-up assessment, usually six weeks after discharge. However, by the time of the most recent assessment, the incidence of paraesthesia, lethargy, balance, reflexes, mood change, tinnitus, headache, co-ordination and urinary problems and weakness had increased. The incidence of cognitive impairment and pain remained unchanged and only the incidence of objective sensory disturbance continued to decrease.

As reported by Sutherland, the incidence of mood disorders, unlike other symptoms, increased over time and was attributed to chronic ill health and inability to return to diving.[3] Cognitive impairment, although present, was not regarded as a major concern by the tuna farm divers, who no longer relied on their memory and avoided situations requiring cognitive skills. The divers thought that lack of external marks decreased people's acceptance of their genuine illness. Many felt that they were regarded as malingerers by the diving community of Port Lincoln.

Hearing was not routinely measured in patients presenting with DCI and none of our subjects complained of hearing loss.

Assessment of gait often consisted of observation of the patient, as he walked to and from the chamber. Subtle changes, requiring walking on inclines or rapid turn around for detection, may have been missed. This may explain the anomaly between the percentage of patients documented as having poor balance or lower limb weakness and normal gait.

Although the previous health of the tuna farm divers was unknown, we can presume given their age and the demands of their occupation, that they were in reasonably good health. The higher incidence of many signs and symptoms amongst the tuna farm divers in comparison to the other diving groups may reflect the way in which data was collected or may represent widespread system involvement. The Divers Alert Network data is based on signs and symptoms reported to them. The experience of the examining physicians is unknown and signs or symptoms may have been overlooked. In contrast, the tuna farm divers were examined by hyperbaric specialists and the medical notes carefully reviewed. The majority of subjects who contributed to Rivera's data were military or commercial divers (96.7%).[2] Ninety-six percent received recompression treatment within 24 hours. Delay to treatment was found to be significantly related to outcome. Although the delay to recompression among recreational divers is unknown, it is likely to be days rather than weeks. In contrast, two thirds of the tuna farm divers waited weeks and months before seeking treatment and continued to dive despite being unwell. This repeated insult and failure to seek prompt medical intervention might account for their higher incidence of most symptoms and signs.

**Long term sequelae**

Pain, cognitive impairment, lethargy, mood swings and paraesthesia were the commonest long-term sequelae, persisting in 25-52% of the tuna farm divers. Divers underwent neuropsychological testing if cognitive impairment was suspected. Table 3 (page 5) lists the tests administered.

The timing of the testing varied from immediately post recompression therapy to seventeen months after presentation. Eight of the twelve divers tested had possible organic impairment and one was significantly impaired. Seven divers had repeat testing between one and two years later. Of these, one diver, with significant impairment on his first evaluation, remained unchanged, five showed improvement and one diver had deteriorated.

All the tuna farm divers suspected of cognitive impairment presented with chronic DCI. Although the other

**TABLE 3**

**NEUROPSYCHOLOGICAL TESTING**

| | | |
|---|---|---|
| Wechsler Adult Intelligence Scale–Revised | E.g. What piece of the picture is missing?<br>E.g. How are two things alike? | Test of psychomotor speed, executive functions and construction. |
| Wechsler Memory Scale-Revised | Recall of newspaper type paragraphs read to subject. | Test of immediate and delayed recall. |
| Rey Auditory Verbal Learning Test | Five presentations with recall and recognition of a fifteen word list. | Measures immediate memory span, short-term and longer-term retention. |
| Rey Complex Figure Test | Immediate and delayed recall of a complex figure. | Test of constructional and memory abilities. |
| Trail Making Test | Part 1: draw lines to connect consecutively numbered circles.<br>Part 11: alternates between consecutively numbered and consecutively lettered circles. | Test of visual conceptual and visuomotor skills. |
| Controlled Oral Word Association Test | Selecting an associated word within certain guidelines. | Test of fluency and self-monitoring. |
| National Adult Reading Test | 50 phonetically irregular words. | Estimate of premorbid mental ability. |
| Hospital Anxiety and Depression Scale | Brief and well standardised self-report. | Measure of anxiety and depression. Excludes somatic symptoms. |
| Neurobehavioral Inventory | Questionnaire completed by subject and relative. | Measure of physical, cognitive and emotional symptoms. |
| Beck Depression Inventory | 21 item scale. | Determines presence and intensity of depression. |
| Spielberg State- Trait Anxiety Inventory | Questionnaire | Measure of trait (temperament) and state (acute) anxiety. |

tuna farm divers had no obvious cognitive problems at follow-up, neuropsychological assessment might have revealed otherwise unrecognised deficits. The sensitivity of such testing is increased when baseline data is available. However, in the absence of such data, repeated assessments for an individual diver may detect subtle deficits in the presence of a normal clinical assessment and can provide a measure of the diver's progress.

### Clinical Scoring System

To describe the severity of decompression illness and to estimate the response to recompression therapy and time, the Royal New Zealand Navy (RNZN) clinical scoring system, designed as a prospective tool, was used.[4]

The RNZN system grades twenty-one signs and symptoms, shown in Table 4, on a scale of 0-3 (none, mild,

**TABLE 4**

**THE TWENTY-ONE SIGNS AND SYMPTOMS USED IN THE ROYAL NEW ZEALAND NAVY CLINICAL SCORING SYSTEM**

| | | |
|---|---|---|
| Lethargy | Cognitive disturbance | Gait |
| Mood change | Visual disturbance | Reflexes |
| Headache | Genitourinary function | Weakness |
| Hearing loss | Bowel dysfunction | Sensory loss |
| Nausea | Co-ordination | Rash |
| Tinnitus | Speech disturbance | Pain |
| Paraesthesia | Lymphatic involvement | Balance |

moderate or severe) based on objective findings for signs and semantic anchors for symptoms.

Each symptom or sign is then weighted by a factor which includes specificity for DCI, natural history if left untreated, potential to incapacitate and co-dependence. Co-dependent symptoms or signs are those that prevent or invalidate the assessment of other symptoms or signs, thereby compromising the severity score. For example, lower limb weakness will interfere or prevent assessment of gait, balance and co-ordination. The dominant symptom or sign, as outlined by the scoring system, is retained and reweighted and the signs and symptoms which may be invalidated are removed. This score is then multiplied by a progression factor, which depends on whether the patient's condition is improving, relapsing remitting or static. A total score was obtained for each assessment.

Most symptoms were easy to grade according to the semantic anchors in the RNZN system. However, pain and fatigue were more difficult to assign a psychometrically sound score to. For pain, the RNZN system converts a visual analogue score of 1-10 to mild, moderate or severe. However, in this group of divers descriptions such as "aches", "stabbing pains", "twinges" etc. were recorded. To grade these descriptions of pain objectively, the descriptions recorded in the notes were listed, and hyperbaric consultants at the RAH were asked to divide them into mild, moderate or severe. Descriptions of fatigue were similarly graded. Signs had been recorded in objective medical terms and therefore easy to grade.

Although the initial history and examination were the most comprehensive, not all the signs and symptoms used in the RNZN system were recorded. Initially only signs and symptoms specifically recorded in the notes were used to determine the severity scores.

The patient's progress was often recorded using such terms as "slight improvement", "much better" or "no real change". However, when severity scores were plotted against time and compared with the impressions recorded, it became apparent that some low scores were the result of certain symptoms and signs not being specifically mentioned on that day, rather than an actual improvement in the patient's condition. To reflect the patient's progress more closely, modifications were required.

Although not mentioned specifically, the presence or absence of some symptoms or signs can be implied from general comments. Remarks such as "no change over last 24 hours" or "feels 100% today" allowed us to attribute a value to certain symptoms or signs, recorded as "implied " in the database.

Occasionally a symptom or sign was recorded on a particular day, not mentioned the next and recorded again the following day. As a result a lower score was calculated for the day in between, even though, judging from the notes, the patient had not improved. Certain features of DCI, such as headache, fatigue, mood or pain may vary from day to day and one cannot presume their presence or absence unless specifically alluded to. However, where balance, gait or co-ordination was recorded as poor on day one and day three with no obvious change on day two, we assumed a similar "interpolated" score for that day.

Scores that include implied and interpolated data were used for analysis as they reflected the clinical situation more accurately.

**TABLE 5**

**SIGNS AND SYMPTOMS AT PRESENTATION AND FOLLOW-UP**

| Signs and Symptoms | Presented with % | First follow-up % | Most recent follow-up % |
|---|---|---|---|
| Patients | n=22 | n=21 | n=21 |
| Pain | 95 | 52 | 52 |
| Paraesthesia | 64 | 10 | 25 |
| Lethargy | 82 | 20 | 30 |
| Cognitive change | 73 | 35 | 35 |
| Objective sensory change | 59 | 5 | 0 |
| Balance | 45 | 15 | 25 |
| Reflexes | 41 | 10 | 15 |
| Headache | 41 | 0 | 10 |
| Mood change | 27 | 15 | 35 |
| Upper limb co-ordination | 27 | 5 | 10 |
| Lower limb weakness | 27 | 0 | 5 |
| Upper limb weakness | 18 | 0 | 5 |
| Dizziness | 9 | 0 | 0 |
| Tinnitus | 9 | 0 | 5 |
| Urinary problems | 4 | 0 | 10 |
| Nausea | 4 | 0 | 0 |
| Lower limb co-ordination | 4 | 0 | 5 |
| Gait | 0 | 5 | 5 |

**Follow up**

Unlike other groups of divers who are often lost to follow-up, 21 of the 22 divers continued to attend the RAH. One diver with residual symptoms flew to Hobart against medical advice. Although he is excluded from the first and most recent follow-up data, his general health and failure to recover from his decompression illness became known to us and he is included in the failure to return to diving data. Signs and symptoms at presentation, first follow up and most recent follow up are shown in Table 5

Although the incidence of all signs and symptoms decreased with recompression therapy, there were significant residual sequelae at the first follow-up visit. Fifty-two percent of the tuna farm divers complained of pain, 35% had some degree of cognitive impairment and 20% reported lethargy. Similar figures have been reported at one month[5] but few hyperbaric units provide details of residual sequelae and it is difficult to estimate whether the figures reported here are unusually high. After the six week assessment, the incidence of pain and cognitive disturbance remained unchanged and the incidence of many other signs and symptoms increased. Pain, cognitive impairment, lethargy and mood swings, the commonest long-term sequelae amongst the tuna farm divers, hindered the securing of alternative employment and contributed to domestic unrest. Mood disorders, the incidence of which increased over time, may have been a direct result of DCI or a reaction to the changes in health, occupation and personal relationships. Not only do many signs and symptoms not resolve with time, but many signs and symptoms return following an initial resolution. Therefore, to assess the efficacy of recompression therapies, longer-term follow-up studies are necessary.

Any diver who returned to diving had done so within ten months of discharge. Seven divers were asymptomatic at discharge and although two had temporary return of symptoms, all had returned to diving by ten months. Fifteen divers were discharged with residual symptoms. The three who returned to diving had done so by four months.

**Comparison of acute and chronic groups**

The tuna farm divers were divided in to two groups, acute and chronic, according to the delay to presentation. Their presentation scores, final scores and degree of recovery are outlined in Table 6. No statistically significant difference was found between the two groups, in presentation scores or final scores, using a two-tailed independent t-test. However, the difference in their final scores may be clinically significant, as the acute group made a statistically significant recovery (p=0.003) and this was reflected in the number of acute divers who were fit to return to diving (4/6 or 67%) compared with the chronic group (6/16 or 37%).

**Assessment of severity**

Results are presented as means ± standard deviations and as means + ranges. Comparisons between groups were made using independent two tailed t-tests, alpha = 0.5. Dependent t-tests were used for intra-group comparisons.

The changes in severity scores over time are presented graphically for groups and individuals. Assessments, which are depicted on the y-axis, are before each recompression treatment, at discharge, at the first follow-up visit, and at yearly intervals thereafter. Severity scores, obtained using the RNZN system, are depicted on the x-axis and are shown with standard deviations where appropriate. Divers were grouped for comparison according to whether their presentation was acute or chronic, whether they were fit or unfit to return to diving, and whether they presented before or after the intervention by WorkCover Corporation.

Some observations can be drawn from the graphs of "severity score and assessments" for all divers and individual divers. A quite dramatic improvement often resulted from the initial RN 62 which then continued at a lesser rate, with some patients becoming symptom free and others reaching a plateau with no further improvement with recompression The response to hyperbaric therapy and the course of the disease thereafter varied considerably between divers.

Figures 2, 3 and 4 depict the severity of DCI and its response to recompression therapy and time for all the divers, the acute group and the chronic groups respectively

**Case reports**

Case 1, who usually did 50-60 ascents a day, developed symptoms after a rapid ascent following compressor failure. He presented on the same day with pain

**TABLE 6**

**PRESENTATION AND FINAL SCORES**

| | Acute (N=6) | | Chronic (N=16) | | P Value |
|---|---|---|---|---|---|
| **Presentation score** | 29.9 | (± 11.2) | 23.2 | (± 14.9) | p=0.601 |
| **Final score** | 3.0 | (± 6.5) | 114.6 | (± 17.6) | p=0.111 |
| **Recovery (Presentation - Final) score** | 16.9 | (± 9.2) | 8.7 | (± 21.0) | |
| **P value of recovery score** | p = 0.003 | | p = 0 133 | | |
| **Return to diving** | 4/6 (66%) | | 6/16 (37%) | | |

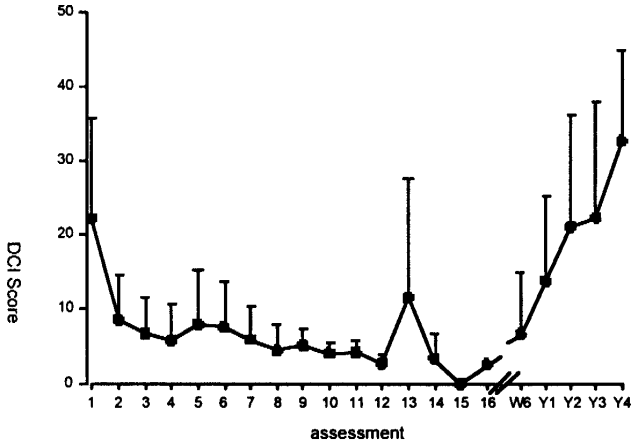


**Figure 2**. The average severity scores+SD for all divers in response to recompression therapy and time. Assessments before recompression treatments (1-16), at the six-week follow-up visit (w6) and yearly thereafter (y1-y4), where applicable, are represented on the x-axis. The severity scores are represented on the y-axis. Where no error bars are shown, n=1.

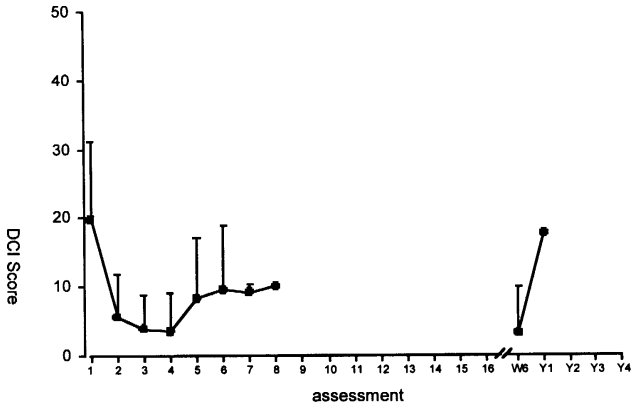


**Figure 3**. The average severity scores +SD over time for those divers who had symptoms for five days or less, i.e. acute decompression illness, before receiving hyperbaric therapy. No error bars are shown where n=1.

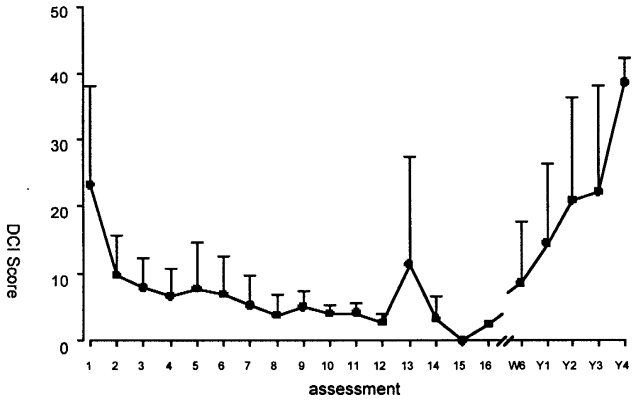


**Figure 4**. The average severity scores over time for those who had symptoms for two weeks or longer, i.e. chronic decompression illness, before receiving hyperbaric therapy. Where no error bars are shown, n=1.

in his knees and back, paraesthesia in his right foot and left hand, lethargy and headache. He had been well until this incident. After five treatments all symptoms resolved (Figure 5) and he returned to diving.

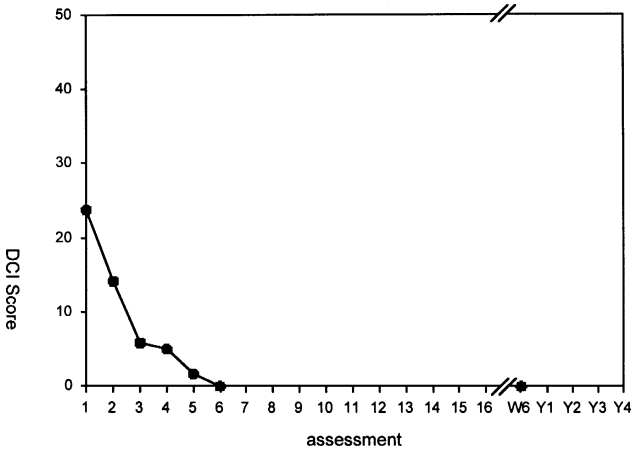


**Figure 5**. Case 1 who had complete resolution of his symptoms with recompression therapy and remained well.

Case 2 became unwell while harvesting at sea, forty eight hours after his last dive. His dive computer showed his most recent dive profiles to be within its no decompression limits. Returning to port took five days and when he reached the RAH, he was vomiting, had paraesthesia in both hands, pain in his arms, legs and chest, lower limb weakness, an abnormal gait, lethargy and cognitive difficulties. He received seven treatments and on discharge still suffered from lethargy, cognitive impairment, difficulties with balance and abnormal gait. All were unchanged at his six week review and the pain in his joints had returned (Figure 6). One year later his symptoms were unchanged. Although unable to return to diving he found alternative work.

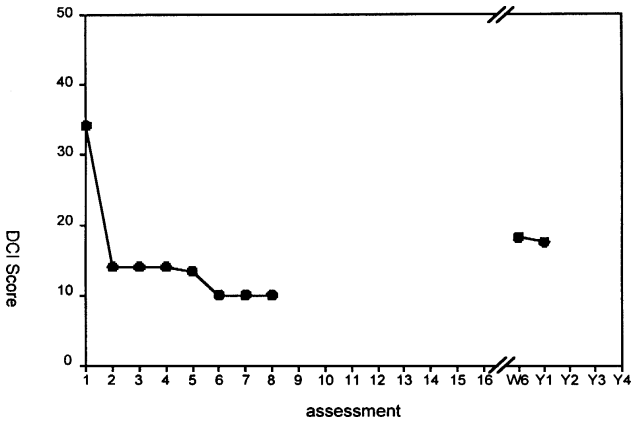


**Figure 6**. Case 2 who had an initial response to recompression therapy and then reached a stage where his symptoms were unaltered by further recompression. At his first follow-up assessment his severity score had increased and his condition remained unchanged one year later.

Case 3 presented to the RAH with a two-week history of aches in his knees and elbows, poor concentration. He was found to have poor co-ordination and impaired balance. He gave a history of 3-4 dives per day with multiple ascents and descents. After six treatments he became asymptomatic but at his first follow-up visit was again found to have poor balance and evidence of cognitive impairment. Neuropsychological evaluation found no evidence of organic impairment. His symptoms resolved over the next six months and although he returned to diving, he chose not to return to tuna farming (Figure 7).

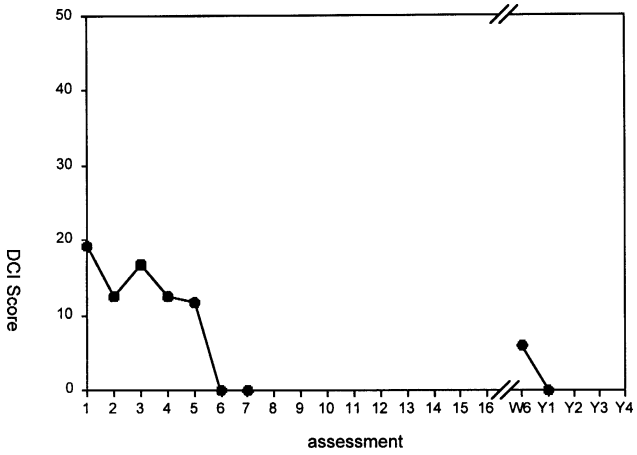


**Figure 7**. Case 3 who had a complete response to recompression therapy, had return of symptoms by the time of his first follow up visit and subsequently became symptom free again.

Case 4 presented with a six-week history of arthralgia, lethargy, headaches, and decreased libido, was found to have objective sensory loss over his arms. He reported diving 3-4 times per day with multiple rapid ascents. At discharge he was complaining of aches in his joints and occasional headaches. Within six months he was symptom free and had returned to diving (Figure 8).

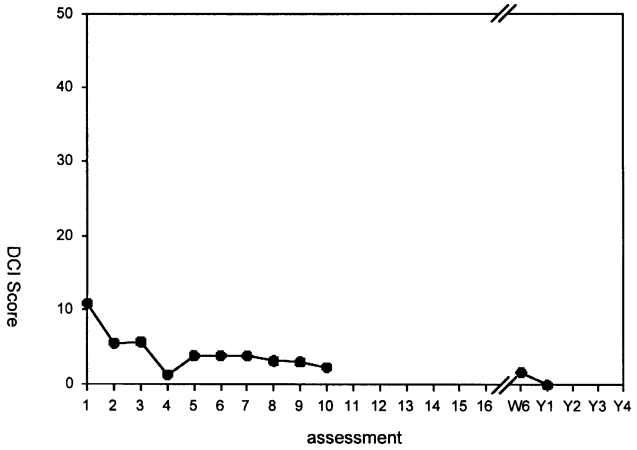


**Figure 8**. Case 4 who was symptomatic on discharge but became asymptomatic with time.

Case 5 had been unwell for 10 weeks, with pain in several joints, intermittent headaches and paraesthesia in both hands, was found to have impaired cognition, abnormal reflexes, poor co-ordination, objective sensory disturbance and difficulties with balance. He received 14 hyperbaric treatments and felt well on discharge. At his first follow-up visit he complained of arthralgia and short-term memory and concentration difficulties and four years later these symptoms persist (Figure 9). He was unable to return to diving but found alternative employment.

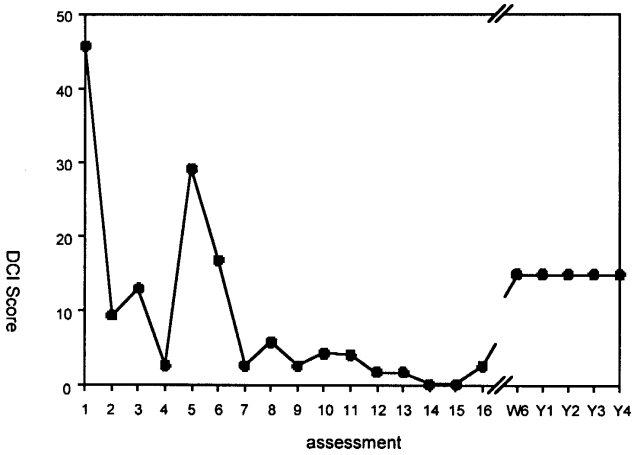


**Figure 9**. Case 5 who had severe decompression illness on presentation, had a fluctuating response to recompression therapy, was almost symptom free on discharge and although he deteriorated afterwards, still maintained a significant degree of his recovery.

Case 6 had been unwell for four weeks with pain in his left shoulder and right hip, intermittent paraesthesiae of both hands, lethargy, headaches and cognitive problems. On examination, he had impaired balance and objective sensory impairment. He received four recompression treatments and intravenous lignocaine and on discharge still had persistent pain, abnormal balance, cognitive impairment and lethargy, although his paraesthesiae had resolved. At his first follow up assessment, all symptoms were still present, his paraesthesiae and objective sensory abnormality had returned and he was anxious and depressed. Subsequent assessments showed impaired co-ordination, abnormal reflexes and decreased strength in all limbs. His neuropsychological assessment revealed possible organic impairment, with deterioration on repeat testing (Figure 10 page 10). He had severe pain necessitating referral to the Chronic Pain Unit at the RAH and was unable to return to any type of work.

**Long term follow up**

Seven divers were symptom free on discharge and all eventually returned to diving. Two of these had return of symptoms after discharge but had become symptom free

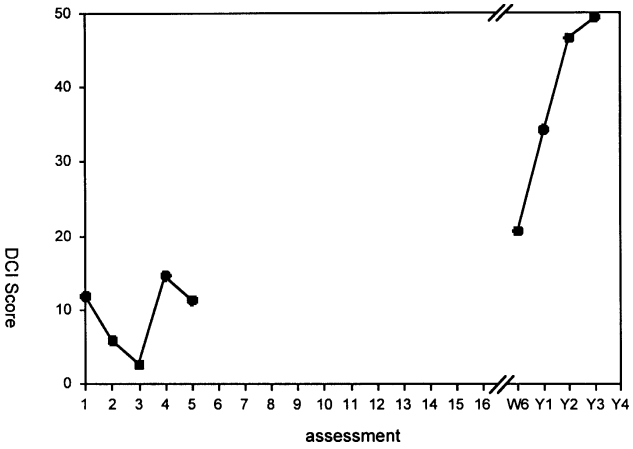


**Figure 10**. Case 6 who had a poor response to recompression therapy and deteriorated further over time.

again by ten months. Fifteen divers had residual symptoms on discharge and only three returned to diving. These three had become symptom free within four months of discharge. All those who returned to diving did so within ten months of discharge.

Approximately two thirds of the divers had residual symptoms on discharge and had received, on average, twice as many recompressions as those who were symptom free (Table 7)

In May 1999 twelve still remained unwell, of these six had found alternative employment, one was seeking a supervisory role, one had returned to study and three divers were unable to work because of severe residual symptoms. The status of the man who went to Hobart is unknown.

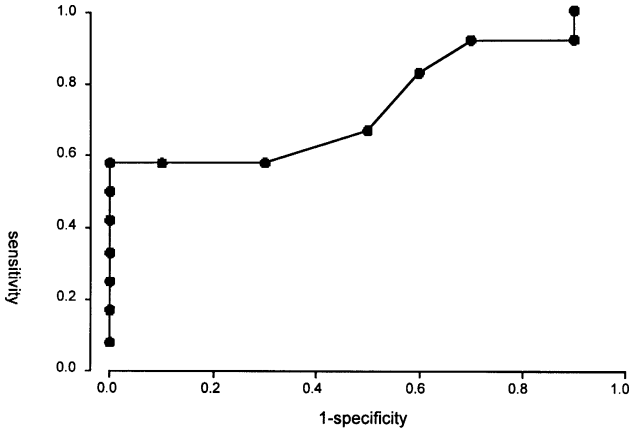


**Figure 11**. Receiver Operating Characteristic Curve based on various hypothetical presentation scores, represented by the black dots, and the calculated sensitivity and specificity of those scores in predicting which of the tuna farm divers would not be fit to return to diving. The point with the highest sensitivity and specificity (lowest 1-specificity) is the score that most accurately predicts which divers will be unfit to return to diving.

**TABLE 7**

**SYMPTOMS ON DISCHARGE AND AVERAGE NUMBER OF TREATMENTS.**

| | Number of divers (%) | Average number of treatments (range) |
|---|---|---|
| Symptom free | 7 (32%) | 4 (2-8) |
| Residual symptons | 15 (66%) | 9 (3-14) |

The RNZN scoring system was used to create a Receiver Operating Characteristic Curve (Figure 11). The best fit is a score of 25 or greater which predicts, with a sensitivity of 58% and a specificity of 90%, that a diver will not be fit to return to diving.

By continuing to assess divers with DCI using the RNZN scoring system, a predictive score with increased sensitivity and specificity may be reached and changes in this predictive severity score may help gauge the efficacy of different treatment regimes.

## Discussion

### LONG-TERM HEALTH OF DIVERS

There is concern about the long-term health of both professional and recreational divers. International conferences held in Luxembourg 1978, Maryland 1981 and Stavanger, Norway 1983 addressed the issues of deep diving and its neuropsychological sequelae without reaching a consensus. Ten years later in Godoysund, Norway the focus shifted from the neurological effects of deep diving to the possible effects of professional diving on a divers health in general. The final statements included "there is evidence that changes in bone, the central nervous system and the lung can be demonstrated in some divers who have not experienced a diving accident or other environmental hazard. The changes are in most cases minor and do not influence the diver's quality of life. However, the changes are of a nature that they may influence the diver's future health".[6] Attention is now focused on two areas: anecdotal reports of cognitive and personality changes in persons exposed to hyperbaric conditions without experiencing overt episodes of decompression illness; and the persistence of signs and symptoms, especially neurological and neuropsychological, after recompression therapy for decompression illness.

In 1959 sequelae, including intellectual impairment, two years after treatment for DCI was reported in eighty six out of 100 caisson workers.[7] Subjective symptoms, such as irritability or headache, were present in the absence of any objective signs and often returned following an initial full recovery.

In 1976 neurological and neuropsychological examinations applied to 10 divers who had suffered DCI involving the central nervous system showed that nine were abnormal on at least one test, while seven were abnormal on both.[8] Similar tests applied to nine divers who had suffered near miss diving accidents, due to air embolus, hypoxia, $CO_2$ poisoning and DCI, led Vaernes and Eidsvik, in 1982, to conclude that a severe diving accident could lead to cerebral dysfunction.[9] The incidence of sequelae following decompression illness amongst military groups has been low and may be related to relatively short delays to treatment.[10-12] Recreational divers do not fare so well. Table 8 (page 12) summarises outcomes following decompression illness in 21 reports over 20 years.[3,5,10-28]

In 1993 a review of 11 Australasian hyperbaric centres revealed that up to 60% of those treated for DCI failed to recover fully following hyperbaric therapy.[29] The residual sequelae reported included depression, impaired cognition, motor and sensory disorders. Even the figures of resolution at discharge may be questionable. In 1988 Curley reported five cases of subtle cognitive impairment following hyperbaric therapy for decompression illness, with abnormalities barely discernible on standard neurological examination.[30] Routine use of neuropsychological testing might reveal an even greater incidence of sequelae.

The failure of decompression illness to resolve completely and its ability to recur after an apparent full recovery is widely accepted. What is less certain is the incidence of subtle residual cognitive changes post-recompression and whether long-term divers are susceptible to neurological or neuropsychological changes in the absence of decompression illness.

Several authors who contributed to a workshop on the effects of deep diving found little cause for concern.[31-33] Other studies have been less reassuring**.** A review of 82 saturation divers revealed more than 10% impairment of intellectual function on repeat testing.[34] As far back as 1976 Black stated that: “20% [of abalone divers] have chronic problems involving ear damage; 10% have suffered some brain losses according to our medical adviser”.[35] A British report claimed that deep-sea diving experience correlated inversely with memory and reasoning skills.[36] While supporting data was lacking in these two studies, they echoed the belief that a dementia or “punch drunk” syndrome existed among occupational divers. Thirty professional abalone divers of New South Wales underwent neuropsychological testing and there was evidence of impairment of acquired intellectual capacity in eleven in the absence of obvious neurological deficits.[37] Another study of abalone divers, with fishermen as a control group, revealed no evidence of cognitive impairment.[38] However, a limited number of neuropsychological tests were used and the level of experience of those applying the tests varied. A postal survey of urchin divers revealed that 18% had chronic medical problems while only 2% gave a history of recompression therapy.[39] However the response rate was only 22% and the symptoms included those of barotrauma as well as DCI.

No consensus has been reached on the probability of long term neurological or psychological deficits in the absence of decompression illness. However, many occupational diving groups stray far from recommended dive practices so health and behavioural changes may simply be the result of unrecognised and untreated episodes of DCI.

## CLINICAL AND NEUROPSYCHOLOGICAL EXAMINATION

No test exists for the diagnosis of DCI. It may occur following a dive within recommended limits for safe diving and in the absence of any known risk factors. Certain signs and symptoms are associated with DCI and the clinical examination is essential to its diagnosis and management. The distribution of symptoms and signs may differ between divers and may vary with treatment and time. Of particular interest are the symptoms or signs that fail to resolve and although many units publish figures on the percentage of divers who fail to make a complete recovery at discharge, residual sequelae and their evolution with time are not well documented.[29] Residual sequelae such as pain or paraesthesia may be easily recognised, whereas sequelae such as mild cognitive impairment or subtle personality changes may be less obvious, and may occur in the absence of any abnormality on standard neurological examination, magnetic resonance imaging or computerised tomographic scanning.[40,41]

Neuropsychology is an applied science concerned with the behavioural expression of brain dysfunction and neuropsychological testing has been proposed as a sensitive marker of cognitive impairment.[30] It has proved useful in determining the effectiveness of recompression therapy but there are limitations.[8,30] Testing requires the co-operation of the subject. The examiner must determine whether the subject attempts the tests to the best of his ability and the presence of depression or anxiety can obscure the existence of organic impairment. These tests have been shown to be valid and reliable in a non-diving population but it is only recently that normative data for various diving groups is being gathered.

## COMPARISON OF FIT AND UNFIT TO RETURN TO DIVING

Table 9 (page 13) compares the presentation scores and response to treatment of those who were able to return to diving and those that remained unwell. As expected, the average final score of those who returned to diving was significantly lower than that of the divers who had

**TABLE 8**

**RESIDUAL SEQUELAE AT DISCHARGE AFTER TREATMENT FOR DCI 1978-1998**

| Year | Author | Subjects | Number | Treatment | Comment | Residual Sequelae |
|---|---|---|---|---|---|---|
| 1978 | Bayne[10] | Military | 50 | USN $O_2$ | One treatment | 0 |
| 1980 | Kizer[13] | Recreational | 157 | USN $O_2$ | Delay>7 hours | 17% |
| 1982 | Kizer[14] | Recreational | 50 | USN O2 | Delay>12 | 34% |
| 1986 | Robertson[15] | Recreational | 28 | USN $O_2$ | 25 DCS<br>3 CAGE | 20%<br>33.3% |
| 1987 | Gorman et al.[16] | Recreational | 87 | USN $O_2$ | Loss of patients to follow-up may have caused bias | 30/46 at one week<br>10/46 at one month |
| 1988 | Gorman et al.[17] | Recreational + Occupational | 64 | USN $O_2$ | 58 DCS<br>6 CAGE<br>Mean delay 26.6 hours | 54.5% DCS<br>33.3% CAGE |
| 1989 | Green et al.[11] | Military | 292 | USN 5<br>USN 6 | Type 1 DCS | 4.1% |
| 1989 | Wirjosemito et al.[12] | Military (Altitude) | 133 | USN $O_2$ | Type 11 DCS | 2.3% |
| 1990 | Bond et al.[18] | Recreational | 347 | Enhanced (165 or 60 fsw +extension)<br>Regular 60 fsw or less | Significant improvement with regular tables | 52%<br>37% |
| 1990 | Walker[19] | Recreational | 50 | USN $O_2$ | | 40% |
| 1990 | Brew et al.[5] | Recreational | 125 | USN $O_2$ | 93 DCS<br>32 CAGE | 60% at discharge<br>51% at one month<br>42% at discharge<br>50% at one month |
| 1991 | Lee et al.[20] | Recreational | 374 | | Mean delay 5.2 hours | 148 |
| 1991 | Weinmann et al.[21] | Recreational | 100 | USN $O_2$ | | 34% |
| 1991 | Todnem et al.[22] | Recreational | 34 | | 18 divers recompressed > 6 hours | 41% |
| 1992 | Acott[23] | Recreational | 20 | USN $O_2$ | | 15% at one month |
| 1993 | Sutherland et al.[3] | Recreational | 25 | USN $O_2$ tables | Questionnaire at 1 year +/-examination | 48% at discharge<br>74% at one year |
| 1993 | Aharon-Peretz et al.[24] | Recreational | 68 | USN $O_2$ Comex 30 | Spinal cord DCI | 21% |
| 1996 | Gardner et al.[25] | Recreational 98% | 100 | USN $O_2$ RNZN Heliox Nitrox or Air/$O_2$ | Mean delay 8 hours ±13.3 | 30% |
| 1996 | Lawler et al.[26] | Recreational | 68 | | Questionnaire at 2 years | 35% |
| 1998 | Francis[27] | Recreational 81% Military 6% | 594 | | | 18% |
| 1998 | Richardson et al.[28] | Recreational 92% | 95 | $O_2$ RNZN heliox Nitrox or Air/$O_2$ | Mean delay 67 hours | 25% |

**TABLE 9**

**SCORES OF THOSE FIT AND UNFIT TO RETURN TO DIVING**

| | Fit to return to diving (10 divers) | | Unfit to return to diving (12 divers) | | P value |
|---|---|---|---|---|---|
| **Presentation score** | 15.7 | (±7.7) | 28.4 | (±15.9) | p=0.033 |
| **Final score** | 0.2 | (±0.5) | 21.3 | (±16.9) | p=0.001 |
| **Recovery score** | 15.5 | (±7.9) | 7.1 | (±24.7) | |
| **P value of recovery score** | p=0.0001 | | p=0.361 | | |

continuing symptoms. However, those who recovered had significantly less severe disease on presentation and their degree of recovery was much higher.

The progress of the disease with treatment and time is represented graphically in Figures 12 and 13.

Although the course of DCI following hyperbaric treatment is uncertain, resolution over a period of years is not supported by this study, where all those who returned to diving had done so by ten months and four divers deteriorated with time. There was considerable variation between divers in their response to recompression and the course of the disease thereafter but the presence or absence of symptoms on discharge was a reasonable predictor of the likelihood of returning to diving.

Although there was no statistically significant difference in presentation or final scores between the acute and chronic groups, their response to treatment differed, with a higher percentage of the acute group returning to diving (67% v 37%). The poor response of the chronic group to recompression therapy may have been due to the delay to treatment or to the fact that this group continued to dive while unwell or both. For many years, interest has surrounded the relationship between delay to treatment and outcome with no clear consensus being reached. Early papers suggest that delay to treatment is an important predictor of outcome,[2,42,43] whereas more recent analyses found no significant correlation.[5,15,17] While one might expect a less successful outcome with delay to treatment, divers more severely affected may present earlier and still, as a consequence of disease severity, recover less fully.[25,29] However the delays in the these studies have been of the order of days, not weeks and months, as was the case with the chronic group of tuna farm divers. Recovery from DCI may be dependent on neuronal recruitment, a process that may be hampered by continuing to dive outside recommended limits.[17] The tuna farm divers, who continued to dive after developing symptoms of DCI, presumably exacerbated the pre-existing inflammatory process and may have hindered the development of compensatory mechanisms.

A good response to treatment may depend on a number of factors including less severe disease at presentation and relatively early presentation or ceasing diving after symptom onset. As the group who had a delayed presentation were also the group that continued to dive, it is not possible to separate the last two factors.

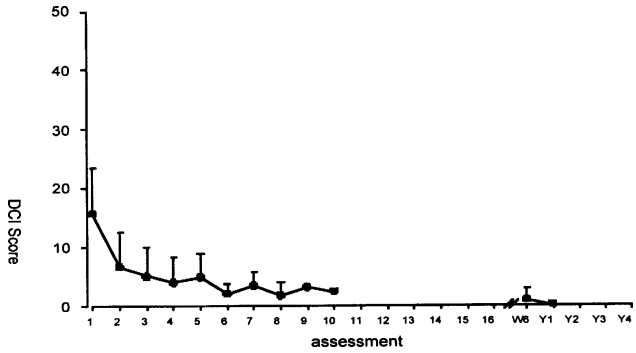


**Figure 12**. The average severity scores+SD over time for those fit to return to diving. Where no error bars are shown, n=1.

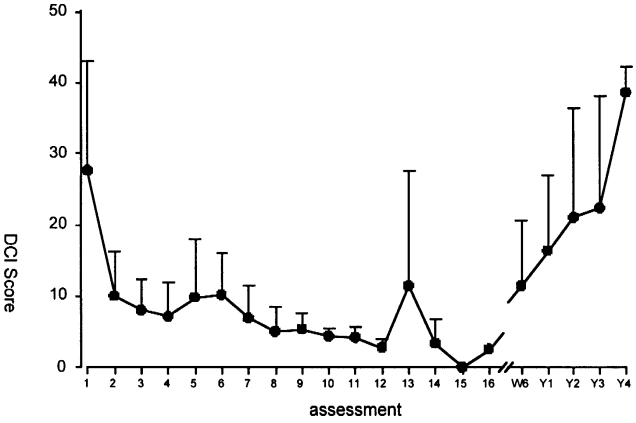


**Figure 13**. The average severity scores+SD over time for those deemed unfit to return to diving. Where no error bars are shown, n=1.

## Conclusions

There was a high incidence of DCI among tuna farm divers, probably due to lack of knowledge of the risks of repetitive diving, of the symptoms of DCI and continuing

to dive when unwell. The incidence has been reduced since intervention by the Department of Industrial Affairs and WorkCover Corporation emphasising safe diving and adequate training of divers. However, although there has been no difference in the severity of DCI at presentation nor in the delay to treatment the divers have made better recoveries and all have returned to some form of employment. This may be the result of fewer ascents and fewer divers now diving outside recommended limits, factors which are known to impede recovery.

Neuropsychological testing suggested the possibility of residual organic impairment among many of the tuna farm divers treated for DCI, but lack of base line data and the presence of depression or anxiety prevented more definitive conclusions. Reports from divers and their partners of changes in memory and concentration, together with the results of neuropsychological testing, suggest the presence of organic impairment after DCI .

The response of the divers to recompression treatment and time varied. Factors such as severe disease at presentation and waiting longer than two weeks before seeking help were found to be associated with a poor outcome. However those divers who waited two weeks or longer before receiving recompression treatment also continued to dive during this time and it is not possible to ascertain which factor was more significant.

The expectation that those with less severe disease on presentation would make a better recovery is supported by this study, where tuna farm divers who were able to return to diving had, on average, significantly lower presentation scores than those who remained unwell.

The incidence of residual sequelae was significant. Fifteen out of 22 divers had residual symptoms at discharge. None of these divers followed for more than a year showed any further recovery and four divers had more severe symptoms, several years after their illness, than on presentation. Twelve divers had long term residual sequelae and were unfit to return to diving and, of these, three had incapacitating symptoms and were unable to return to any form of work.

The RNZN scoring system[4] provided a useful index of disease severity and recovery, showing the variable response of DCI to treatment and the unpredictable nature of the disease over time, and facilitating comparison of subgroups and assessment of interventional strategies.

The inclusion of “implied” and “interpolated” data was felt to reflect the clinical situation more accurately and although it statistically altered the severity score ($p=0.000003$, $DF=238$), the product-moment correlation was 0.99. In other words, while the absolute figures were altered, the trends in the scoring system over time were similar.

A discussion of the effects of the intervention by the WorkCover Corporation and the Department of Industrial Affairs on the incidence, severity and sequelae after treatment of the tuna divers discussed in this paper has recently been published.[44]

**References**


1 Elliott DH and Moon RE. Manifestations of the decompression disorders. In *The Physiology and Medicine of Diving. (4th Ed)*. Bennet PB and Elliott DH. Eds. London: WB Saunders Ltd, 1993: 481-505

2 Rivera JC. Decompression sickness among divers: an analysis of 935 Cases. *Military Medicine* 1964; 129: 313-334

3 Sutherland A, Veale A and Gorman DF. Neuropsychological problems in 25 recreational divers one year after treatment for decompression illness. *SPUMS J* 1993; 23 (1): 7-9

4 Mitchell S, Holley T and Gorman DF. A new system for scoring severity and measuring recovery in decompression illness. *SPUMS J* 1988; 18 (2): 84-94

5 Brew S, Kenny C, Webb R and Gorman D. The outcome of 125 divers with dysbaric illness treated by recompression at HMNZS Philomel. *SPUMS J* 1990; 20 (4): 226-230

6 Godoysund Conference

7 Rozsahegyi I. Late consequences of the neurological forms of decompression sickness. *Br J Ind Med* 1959; 16: 311-317

8 Peters BH, Levin HS and Kelly PJ. Neurologic and psychologic manifestations of decompression illness in divers. *Neurology* 1976; 26: 381-382

9 Vaernes RJ and Eidsvik S. Central nervous dysfunction after near miss accidents in diving. *Aviat Space Environ Med* 1982; 53: 803-807

10 Bayne C.G. Acute decompression sickness: 50 cases. *JACEP* 1978; 7: 351-354

11 Green JW, Tichenor J and Curley MD. Treatment of type 1 decompression sickness using the U.S. Navy treatment algorithm. *Undersea Biomed Res* 1989; 16 (6): 465-470

12 Wirjosemito SA, Touhey JE and Workman WT. Type 11 altitude decompression sickness: U.S. Air Force experience with 133 cases. *Aviat Space Environ Med* 1989 March; (60) 3: 256-262

13 Kizer KW. Dysbarism in paradise. *Hawaii Med J* 1980; 39: 109-116

14 Kizer KW. Delayed treatment of dysbarism. *JAMA* 1982; 247: 255-258

15 Robertson A. Treatment and results of thirty hyperbaric cases at the recompression facility HMAS Stirling. *SPUMS J 1986*; 16 (4): 141-143

16 Gorman DF, Edmonds CW and Parsons DW. Neurologic sequelae of decompression sickness: A clinical report. In *Underwater and Hyperbaric*

*Physiology IX*. Bove AA, Bachrach AJ and Greenbaum LJ Jnr. Eds. Bethesda: Undersea and Hyperbaric Medical Society 1987: 993-998

17 Gorman DF, Pearce A and Webb RK. Dysbaric illness treated at the Royal Adelaide Hospital 1987: a factorial analysis. *SPUMS J* 1988; 18(3): 95-102

18 Bond JG, Moon RE and Morris DL. Initial table treatment of decompression sickness and arterial gas embolism. *Aviat Space Environ Med* 1990; 61 (8): 738-743

19 Walker R. Fifty divers with dysbaric illness seen at Townsville General Hospital during 1990. *SPUMS J* 1992; 22(2): 66-71

20 Lee HC, Niu KC, Chen SH, Chang LP, Huang KL, Tsai JD and Chen LS. Therapeutic effects of different tables on type 11 decompression sickness. *J Hyperbaric Med* 1991; 6: 11-17

21 Weinmann M, Tuxen D, Scheinkestel C and Millar I. Decompression illness: 18 months experience at the Alfred Hospital Hyperbaric Unit 1990. *SPUMS J* 1992; 21 (3): 135-143

22 Todnem K, Eidsvik S and Hjelle J. Neurological decompression sickness. *Tidsskrift for Den Norske Laegeforening* 1991; 111 (17): 2091-2094

23 Acott C. Clinical review: Royal Adelaide Hospital Hyperbaric Unit 1990. *SPUMS J* 1992; 22(1): 51-54

24 Aharon-Peretz J, Adir Y, Gordon CR, Kol S, Gal N and Melamed Y. Spinal cord decompression sickness in sport diving. *Arch Neurol* 1993; 50(7): 753-756

25 Gardner M, Forbes C and Mitchell S. One hundred divers with DCI treated in New Zealand during 1995. *SPUMS J* 1996; 62(4): 222-225

26 Lawler WL, Hargasser S, Moon RE, Uguccioni DM and Stolp BW. Two year follow-up of decompression illness. *Undersea Hyper Med* 1996; 23s: 35

27 Francis J. Decompression illness in sports divers: The UK experience. *SPUMS J* 1998: 28(1): 42-44

28 Richardson K, Mitchell S, Davis M and Richards M. Decompression illness in New Zealand divers: the 1996 experience. *SPUMS J* 1998; 28(1): 50-55

29 Gorman D and Harden M. Outcome after treatment for decompression illness in Australasia. *SPUMS J* 1993; 23(3): 165-168

30 Curley MD, Schwartz HJ and Zwingleberg K. Neuropsychologic effects of cerebral decompression sickness and gas embolism. *Undersea Biomed Res* 1988; 16: 223-226

31 Aarli J. Neurological consequences of deep diving: some case studies. In *Long -term consequences of deep diving.* Shields TG, Minsaas B, Elliott DH and McCallum RI. Eds. Stavanger, Norway: A.s Verbum, 1983; 53-68

32 Thalmann E D. US Navy experience from deep diving. In *Long -term consequences of deep diving.* Shields TG, Minsaas B, Elliott DH and McCallum RI. Eds. Stavanger, Norway: A.s Verbum, 1983; 87-101

33 Torok Z. Recent British simulated deep diving experience. In *Long -term consequences of deep diving.* Shields TG, Minsaas B, Elliott DH and McCallum RI. Eds. Stavanger, Norway: A.s Verbum, 1983; 103-109

34 Vaernes RJ, Klove H and Ellertsen B. Neuropsychologic effects of saturation diving. *Undersea Biomed Res* 1989; 16: 233-251

35 Black JC. Abalone divers require special consideration. *Australian Fisheries* 1976; 35 (1): 20-21

36 Smyth E. Deep-sea diving may cause loss of memory. *New Scientist* 1985; 105-108

37 Edmonds C and Boughton J. Intellectual deterioration with excessive diving (punch-drunk divers) *Undersea Biomed Res* 1985; 12(3): 321-326

38 Andrews G, Holt P, Edmonds C, Lowry C, Cistulli P, McKay B, Misra S and Sutton G. Does non-clinical decompression stress lead to brain damage in abalone divers? *Med J Aust* 1986; 144: 399-401.

39 Butler WP Maine's urchin diver: a survey of diving experience, medical problems, and diving-related symptoms. *Undersea Hyper Med* 1995; 22(3): 307-313

40 Warren LP, Anthony DL, Burger PL, Camporesi EM, Djan WT, Heinz ER, Massey EW, Moon RE and Sallee DS. Neuroimaging of scuba diving injuries to the CNS. *Am J Roentgenology* 1988; 151:1003-1008

41 Hodgson M, Beran RG and Shirtley G. The role of computed tomography in the assessment of neurologic sequelae of decompression sickness. *Arch Neurol* 1988; 45:1033-1035

42 Keays FL. Compressed air illness with a report of a 3,692 cases. *Researches from the Department Medicine, Publications of Cornell University Medical College,* 1909; 2: 1-55.

43 Paton WDM and Walder DN. *Compressed air illness-An investigation during the construction of the Tyne tunnel, 1948-1950. Medical Research Council Special Report Series No. 281.* London, 1954

44 Whyte P, Doolette DT, Gorman DF and Craig DS. Positive reform of tuna farm divers in South Australia in rsponse to a government intervention. *Occ Environ Med* 2001; 58:124-128

*The above paper is an abridged version of the thesis presented by Dr Pauline Whyte MB, BCH, BAO, FFARCSI, Dip DHM, for the Diploma in Diving and Hyperbaric Medicine, awarded to her in July 2000 for work done as an Anaesthetic Fellow in the Hyperbaric Medicine Unit at the Royal Adelaide Hospital.*

*At present Dr Whyte is working as a Senior*

*Registrar in the Department of Intensive Care at the Flinders Medical Centre, Adelaide, South Australia.*

*Dr Whyte's address is 25 Woodhouse Crescent, Wattle Park, South Australia 5066. E-mail <docwhyte@hotmail.com>.*

## EFFECTS OF HYPERBARIC OXYGEN TREATMENT ON BLOOD SUGAR LEVELS AND INSULIN LEVELS IN DIABETICS

Lalith Ekanayake and David J Doolette

**Key Words**

Diabetes, hyperbaric oxygen, hyperbaric research.

**Summary**

Hyperbaric oxygen ($HBO_2$) is commonly used to treat non-healing wounds in diabetic patients. Although anecdotal evidence from hyperbaric centres suggests that diabetics are vulnerable to hypoglycaemia when they are treated with hyperbaric oxygen, there has been little definitive human or animal research showing the effects of hyperbaric oxygen on blood sugar and insulin levels. Blood sugar levels and insulin levels in five diabetic and five non-diabetic subjects were measured both while breathing normobaric air and hyperbaric oxygen . Mean blood sugar levels decreased significantly by $3.5 \pm 0.7$ mmol/l during hyperbaric oxygen breathing in the five diabetics. Insulin dosage was not changed in either condition.

### Introduction

Apart from treatment for diving related illness, hyperbaric oxygen ($HBO_2$) treatment has therapeutic value in many illnesses including non-healing wounds in diabetics. The use of hyperbaric oxygen treatment for diabetes (but not wounds) has its origins in the 1920s with the American physician Dr Orval J Cunningham, who used hyperbaric oxygen for the treatment of various illnesses (syphilis, pernicious anaemia, and cancer) including diabetes mellitus.[1] Anecdotal evidence from hyperbaric centres shows that diabetics are prone to develop hypoglycaemia when they are exposed to $HBO_2$.

Blood sugar levels (BSL) decreased in some[2] or all[3] insulin dependent diabetics after $HBO_2$ treatment. Insulin requirements are reduced during $HBO_2$.[4] Some additional evidence from underwater diving indicates that the long and short-term insulin requirements of diabetics decreased over a period of 7 days of diving.[5]

Although there is disagreement in the diving medical fraternity, a majority of experts believe that diabetes, mainly insulin dependent diabetes mellitus (IDDM), is a contraindication to recreational and commercial diving because hypoglycaemic signs and symptoms may be confused with other diving maladies, hypoglycaemia can cause unconscious underwater and there may be increased likelihood of decompression illness (DCI) in diabetics.[6-9]

The mechanisms of hypoglycaemia during $HBO_2$ treatment are unknown, but it has been postulated[10,11] that $HBO_2$ might:

1 increase tissue oxygen and increase aerobic metabolic energy generation (oxidative phosphorylation), driving up glucose consumption;
2 increase aerobic metabolism in the pancreatic Islets of Langerhans which may stimulate insulin secretion;
3 inhibit the actions of anti-insulin hormones (somatotropin and glucagon); or
4 increase tissue sensitivity to insulin.

BSL have not been previously reported for non-diabetics during $HBO_2$ treatment. However, if hypoglycaemia occurs during $HBO_2$ in diabetics but not in non-diabetics, this may result from failure, in diabetics, of the normal protective mechanism. For instance in non-diabetics during exercise, BSL is maintained by decrease in insulin and rise in glucagon and catecholamine levels. These mechanisms fail in some diabetics, mainly in IDDM patients, resulting in hypoglycaemia.[12]

This study investigates BSL and insulin in diabetics and non-diabetics during $HBO_2$ and normobaric air breathing. The specific hypotheses tested are;
1 $HBO_2$ increases insulin in diabetics but not in non-diabetics and
2 BSL decrease is a result of increase in insulin.

### Method

This study was approved by the Research Ethics Committee of the Royal Adelaide Hospital and was conducted in accord with the National Statement on Ethical Conduct in Research Involving Humans.

### Subjects

Five diabetics (3 males and 2 females) gave their informed consent to participate in this study. Four of them received $HBO_2$ for diabetic foot ulcers and the other for osteoradionecrosis. Mean age of the diabetics was 60 years (range 46–86 years). There were 4 IDDM and 1 Non-insulin Dependent Diabetes Mellitus (NIDDM). The mean diabetic duration was 22 years (range 7-40 years).