ORIGINAL PAPERS

ONE HUNDRED DIVERS WITH DCI TREATED IN NEW ZEALAND DURING 1995

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Key Words

Decompression illness, hyperbaric oxygen, tables, treatment, treatment sequelae.

Introduction

The 1995 calender year was the busiest in the history of the Royal New Zealand Navy Hospital with 100 cases of decompression illness (DCI) treated at the Slark Hyperbaric Unit (SHU). This contrasts with 24 cases in 1990, 31 in 1991, 55 in 1992, 68 in 1993 and 48 in 1994. Demographic data describing this patient population is presented in the following report.

Methods

Relevant parameters pertaining to each case were recorded on a Microsoft Access 2 database maintained by SHU staff. In most cases data entry occurred during the patient's admission, although some records were obtained retrospectively from the patient notes. In selection of data fields, details of the diving incident leading to DCI were not emphasised since these data are forwarded to the Diving Incident Monitoring Study (DIMS) at Adelaide and will be reported elsewhere.

Age and gender of divers

The age of divers ranged from 17 to 64 years, with a mean of 34.0 (SD ± 9.7). Ten divers were female (17 to 49 years, mean 30.5) and 90 male (17 to 64 years, mean 34.5).

Past diving history

Only six divers confirmed that they had received no instruction from a recognised diver training agency. Five divers suffered DCI resulting from entry level training dives. All other divers held recognised diving qualifications although the level of qualification was not recorded in seven. The divers are grouped according to the highest diving qualification held in Table 1 and according to the agency from which they received that training in Table 2.

TABLE 1.

HIGHEST DIVING QUALIFICATION HELD BY DCI PATIENTS.

Highest diving qualification	
Open water diver	40
Advanced open water diver	18
Rescue diver	5
Divemaster	6
Instructor	5
Commercial (HSE Part I)	1
Under training (open water)	3
Under training (resort course)	1
Under training (Navy)	1
Trained but qualification level unknown	7
No qualification or formal training	6

TABLE 2

TRAINING AGENCY PROVIDING HIGHEST QUALIFICATION

PADI	65
CMAS	9
NAUI	5
SSI	5
RNZN	1
US Navy	1
Commercial	1
No qualification or formal training	6
Training history not recorded	7

The number of dives before the dive resulting in DCI ranged from 0 to 4,500 (mean 366 SD ±707). The percentages of divers with less than 20 and 100 dives before their episode of DCI were 40% and 62% respectively. The mean previous deepest depth before the episode of DCI was 20 m.

Seasonal incidence

The peak incidence of DCI was in the summer months of January and February. Numbers of DCI cases are shown by month in Figure 1.

Nature and location of diving

All divers were air diving using scuba equipment with the exception of one diver using a "hookah" (surface supply from a compressor) and one making a chamber dive.

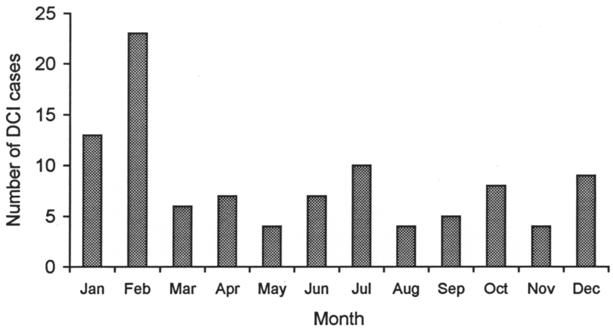

Figure 1. Monthly numbers of DCI patients in 1995.

Ninety were diving for recreation, and 10 for commercial purposes.

Eighteen divers used no recognised dive table or computer. Dive computers were used by 21 divers, and dive tables were used by 52. The means of assessing decompression status, if any, was not recorded for nine divers. The divers are grouped according to the method of decompression status assessment employed in Table 3. Analysis of the reported dive profiles preceding the episode of DCI showed that 39% of divers using dive tables complied with the limits of the table used, whereas 24% of the reported profiles complied with the limits of the Canadian Defence and Civil Institute of Environmental Medicine (DCIEM) table.[1]

Seventy six divers had been diving in New Zealand's North Island, with only 15 in the South Island and nine in the South Pacific Islands.

Referral and transport to the SHU

Divers were usually referred to the New Zealand Diver Emergency Service (DES), based at the SHU, by a local doctor, a local hospital, or by themselves. The frequencies of the various means of referral are given in Table 4. Eighty one of the divers were evacuated, or presented, from the North Island, 13 from the South Island, and six from the Pacific Islands. The frequencies of use of the various means of transport to the unit are given in Table 5. The time from surfacing after the last dive to arrival at the SHU ranged from less than one hour to 28 days (mean 68 hours, SD ±99.6).

Presentation of DCI

The delay to symptom onset varied from immediately on surfacing to 72 hours after diving (mean 8 hours, SD 13.3). Musculoskeletal pain was the most commonly reported symptom. Forty five divers had objective signs when assessed at the SHU. The percentage incidence of the various symptoms and signs recorded is given in Table 6.

TABLE 3

ASSESSMENT OF DECOMPRESSION STATUS

PADI Recreational Dive Planner	40
Computer	21
DCIEM Air Diving Table	6
US Navy Air Diving Table	5
Bassett Modified US Navy	1
No decompression status assessment	18
Decompression assessment not recorded	9

TABLE 4

SOURCE OF REFERRAL TO DES

Local doctor	36
Hospital	29
Self referral	28
Dive boat operator	3
Ambulance service	2
Dive shop	1
Local police	1

TABLE 5

MEANS OF TRANSPORT TO THE SHU

Private vehicle	37
Fixed wing 1 bar pressurised aircraft	30
Road ambulance	17
Helicopter ambulance	13
Not recorded	3

Treatment

Fifty five divers were recompressed to a maximum pressure of 2.8 bar (equivalent to a depth of 18 m of sea water) according to either a USN Table 6[2] (Royal Navy Table 62) or a Royal New Zealand Navy Table 62A,[3] the latter being an oxygen-helium treatment table of similar decompression pattern and duration to the USN Table 6. The remaining divers received deeper treatments after inadequate symptom resolution during initial treatment at 2.8 bar (18 msw). The frequencies of use of the various initial treatment tables are given in Table 7.

TABLE 6

FREQUENCY OF PRESENTING SYMPTOMS AND SIGNS IN 100 DCI PATIENTS.

Symptoms and signs	%
Pain	66
Fatigue	51
Numbness	51
Tingling	46
Weakness	42
Headache	38
Dizziness	23
Shortness of breath	22
Difficulty walking	21
Cognitive difficulty	18
Visual changes	8
Urinary impairment	5
Cough	5
Itch	4
Loss of consciousness	4
Rash	2
Other	15

TABLE 7

RECOMPRESSION TREATMENT TABLES USED AT THE SHU IN 1995

Treatment table	Maximum depth	Treatment gas	No.	Extended treatments
RN 62 (USN 6)	18 msw	O_2	44	10
RNZN 62A	18 msw	Heliox	11	1
RNZN 1	30 msw	Nitrox/O_2	3	1
RNZN 1A	30 msw	Heliox/O_2	8	0
RNZN 2A	30 msw	Heliox	17	12
RN 63 (USN 3)	50 msw	Air/O_2	5	1
RNZN 63	50 msw	Heliox/O_2	12	5

Daily retreatment with an 18:60:30 table[4] was given until the patient either made a full recovery or experienced no sustained improvement over two consecutive days. The mean number of retreatments was 2.3 (SD ±2.7, range 0-17).

Two divers suffered central nervous system oxygen toxicity manifest as convulsions. In one, the episode occurred 10 minutes into the first oxygen breathing period of a USN Table 6 (RN 62). Post-ictally, this patient was changed to 50:50 oxygen-helium and completed an uncomplicated RNZN 62A. In the second, the episode occurred during the third oxygen breathing period of a USN Table 6. This patient completed the table after a recovery period off oxygen. One diver suffered symptomatic pulmonary oxygen toxicity manifested as mild retrosternal discomfort and cough. These symptoms arose near the end of a maximally extended USN Table 6.

Outcome

Seventy divers were recorded as fully recovered at discharge from the SHU while 30 were discharged with residual symptoms or signs. These groups are compared with respect to age, gender, delay to presentation and compliance with dive tables in Table 8.

Discussion

The record 1995 case load was partly explained by the closure of the hyperbaric unit at Christchurch (South Island) throughout the year, making the SHU New Zealand's only hyperbaric unit. Nevertheless, even if the 13 divers evacuated from the South Island are ignored, the remaining 87 divers still represent an annual record. It is possible that particularly good summer diving conditions and New Zealand's economic recovery led to a substantial increase in diving activity and a consequent increase in the number of accidents.

The largest group of patients was relatively inexperienced divers trained to Professional Association of Diving Instructors (PADI) Open Water level,[5] who were using the PADI Recreational Dive Planner (RDP)[6] to assess decompression status. PADI has been the dominant training agency in New Zealand since 1986 and currently trains 70-80% of new divers. It follows that the patient population reported here is probably representative of the active diving population and nothing can be concluded from such numerator based data. Similarly, without an accurate assessment of the prevalence of diving computers in the diving community, little can be concluded from the control of decompression status by these devices in 21 % of the cases reported here.

Although the reported dive profiles may not be reliable, it is notable that 39 % of patients reported dives within the limits of the table used, and 24 % within the limits of the DCIEM tables. The occurrence of DCI despite adherence to the limits of tables is worthy of emphasis in the training of recreational divers.

The presenting symptoms and signs in these patients were qualitatively and quantitatively similar to those reported in other series.[7] The mean delay to symptom onset after diving was 8 hours, while the mean delay to presentation at the SHU after diving was 68 hours. Although the latter figure may be skewed by the very late presenting patients, there is nevertheless a clear tendency for divers to

TABLE 8

COMPARISON OF PATIENTS MAKING COMPLETE AND INCOMPLETE RECOVERY WITH RESPECT TO AGE, GENDER, DELAY TO PRESENTATION AND COMPLIANCE WITH DIVE TABLES

Variable	Complete recovery	Incomplete recovery
Number	70	30
Age (years)	mean 33.7 (SD ±9.5)	mean 36.0 (SD ±10.4)
Gender		
female (n = 10)	8 (80 % of females)	2 (20 % of females)
male (n = 90)	62 (69 % of males)	28 (31 % of males)
Delay from dive to arrival at SHU (hours)	mean 76 (SD ±109) range 0.5 - 692	mean 48 (SD ±68) range 0.5 - 336
Compliance with tables		
Own	43 %	30 %
DCIEM	29 %	13 %

tolerate, or deny, the often non-specific and subjectively mild symptoms of DCI for a substantial period before seeking help, perhaps in the hope of avoiding the stigma and inconvenience of the diagnosis. Such procrastination should be actively discouraged in recreational diver education.

The selection of recompression treatment tables during 1995 was based in part on the algorithm for the trial of oxygen-helium (heliox) versus oxygen in the treatment of air diving DCI (the heliox trial"),[8] although a number of divers were not entered into this trial. The large group of divers treated deeper than 18 m (2.8 bar) utilising oxygen-helium tables (Table 7) includes both divers treated with oxygen and divers treated with oxygen-helium during the initial period at 2.8 bar (18 m). The relative merits of oxygen or oxygen-helium as the initial treatment gas at 2.8 bar (18 m) cannot be implied from these data. These issues will be clarified when the heliox trial is reported. The approach to initial treatment table selection by Australasian hyperbaric units has been reported elsewhere.[4]

Thirty divers were discharged with residual manifestations of DCI. This is a similar proportion of treatment failures to that reported for Australasian sport diver populations elsewhere[9,10] and underscores that there is no room for complacency about the adequacy of current treatment protocols.[11] There was no difference in the mean age of the complete and incomplete recovery groups or the relative proportions of males and females across these groups. The mean delay to presentation in the complete recovery group was 76 hours, compared with 48 hours in the incomplete recovery group. The inference that earlier presentation predicates a poor outcome is untenable and these data probably reflect earlier presentation of divers with more severe disease. Not surprisingly, divers making a complete recovery were more likely to have complied with the limits of the DCIEM table and/or their own dive table than divers who made an incomplete recovery. These data reflect the likelihood of disease of greater severity following a more provocative time/depth exposure.

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AN UNUSUAL CASE OF CEREBRAL ARTERIAL GAS EMBOLISM

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Abstract

A snorkel diver suffered a cerebral arterial gas embolism after breathing, at depth, from the octopus regulator of a scuba diver. The neurological injury was manifested by loss of consciousness and by cortical blindness. The chest X-ray demonstrated multiple signs of pulmonary barotrauma. An MRI scan demonstrated cerebellar infarction. The unusual aspects of this case and the pathophysiology of pulmonary barotrauma are discussed.

Key Words

Accident, air embolism, barotrauma, cerebral arterial gas embolism, eyes, pulmonary barotrauma, treatment, unconscious.

Introduction

Pulmonary barotrauma and cerebral arterial gas embolism (CAGE) are usually associated with scuba diving accidents. We describe CAGE in a snorkel diver demonstrating a number of unusual features.

Case Report

A previously fit 23 year old male was snorkel diving on the Great Barrier Reef. His snorkel diving experience is unknown. He had never dived with scuba. He dived to a depth of 3-5 m and took several breaths from the octopus regulator of an accompanying scuba diver. Then he made an uncontrolled ascent to the surface.

On reaching the surface he was unconscious and had to be rescued. In the boat he was seen to be pale, tachycardic and tachypnoeic. CAGE was immediately suspected, so he was placed supine. Oxygen was given at a flow rate of 15 l/min using a non-rebreathing mask. After 45 minutes the flow was reduced to 10 l/min. He rapidly recovered consciousness, but appeared to be almost completely blind. No formal assessment of vision was carried out on the boat and the patient had no subjective improvement in his vision before recompression. He had no other symptoms or signs.

Urgent helicopter transfer to the local base hospital was arranged. Examination in hospital, an hour and 50 minutes after surfacing and 30 minutes after leaving the boat by helicopter, demonstrated he had difficulty in recognising shapes but normal visual fields to confrontation. This was unusual as occipital blindness usually, but not always, shows loss of field before loss of acuity. Ocular movements were normal, with both pupils small and reacting directly and consensually to light. Fundoscopic examination showed injected fundi but normal discs and no visible haemorrhages. A chest X-ray demonstrated mediastinal emphysema, subcutaneous emphysema and bilateral pneumothoraces (Fig 1).

These findings were confirmed following transfer to Townsville General Hospital 6 hours after surfacing. An ophthalmological opinion was obtained and the visual changes were considered to be the result of bilateral central retinal artery (CRA) occlusions.

He was initially managed with IV saline, IV lignocaine, a Comex 30 heliox table and bilateral digital ocular massage. He was compressed 6.5 hours after surfacing. His vision had improved considerably 85 minutes into the table, but deteriorated on ascent to 12 m.

He was taken back to 30 m and the table recommenced. After this extra time at depth improvement was sustained on ascent. After his first treatment his vision was 6/9 in both eyes.

The fundi were noted to be normal the following day. He had 3 further hyperbaric oxygen treatments (18 m for