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## A CASE OF RECURRENT DECOMPRESSION ILLNESS

Peter Chapman-Smith

**Key Words**

Case report, decompression illness, sequelae, treatment.

General practitioners see their patients repeatedly. This puts them in an excellent position for follow up studies on divers who have suffered decompression illness (DCI) to discover what the usual clinical progress is likely to be. Very little has been published about the long term follow up of divers. Case 1 is from my records. Follow up of divers suffering decompression illness treated with recompression is often revealing.

### Case 1

A 50 year old mechanic has been diagnosed as DCI on 4 separate occasions. His only other disability has been symptoms of carpal tunnel syndrome. He was treated at the Royal New Zealand Navy (RNZN) Hospital on 3 occasions between 1988 and 1996. He has suffered from a series of subtle but significant disabilities for years.

December 1988

After an evening of moderate to heavy alcohol consumption he did a single dive to 21 m (70 ft) for 60 mins. He ran out of air and made a rapid ascent. 24 hours later he consulted me complaining of skin itch, pain in his hands and feeling very tired and light headed. He had pain at the base of his spine and in the buttocks.

Physical examination was neurologically normal, except for a sharpened Romberg Test (SRT) of 25 seconds. He was slow counting down from 100 by sevens. The audiogram showed a mild high frequency loss R>L.

He was transferred to the RNZN recompression chamber (RCC) at the Naval Hospital in Auckland, about 150 km, where he needed three treatments before his

symptoms resolved. Twenty four hours after his third treatment he still had no symptoms. The discharge diagnosis was Type I DCS with skin and joint involvement.

Unfortunately skin itch, anterior chest tightness and tiredness returned two days later. He was unwilling to go for further treatment. He was advised not to dive for 2-3 months.

He then did 10 uneventful dives before the next problem.

January 1990

He did a single dive, for crayfish, to 21-24 m (70-80 ft) for 35 minutes with a 3 minute stop at 4.5 m (15 ft).

Several hours later he developed a generalised skin itch. Three days later he woke with a dull ache (pain) in his left hip. He developed pain in the left shoulder, left elbow, left wrist and in the knuckles of his left hand later the same day. No joint swelling was noted.

Next day he had reduced sensation over the lateral side the left lower leg and dorsum of the left foot. These changes were only noticed by chance. When there had been no change after 36 hours he contacted the RNZN Hospital directly.

He arrived at the Naval Hospital 6 days after the dive. There had been some improvement in his symptoms and the area of numbness was now hyperaesthestic. No urinary, balance or muscle weakness problems were noted. Physical examination showed no abnormalities. Serial sevens were managed easily and the SRT was normal.

It was decided to recompress him as his symptoms were the same as at the time of his first treatment 12 months earlier. He was given a USN Table 6 with total resolution of symptoms at 18 m. He reported clearer thinking after treatment.

Two days later he telephoned me from home to report all was well.

January 1993

He did his first and only dive since January 1990 to a maximum of 18 m (60 ft) for 45 minutes, in excellent weather, after crayfish. On the boat he was exposed to diesel conditioner containing a biocide similar to nerve gas.

After an Automobile Association call out, and several beers, he developed a moderately severe headache 2-3 hours after the dive. Two days later he saw me as the symptoms felt like his previous DCI. He had slow mentation and short term memory with poor balance, so he was referred to the Naval Hospital where he was recompressed repeatedly, the treatments lasting for 2 weeks.

On discharge he was much better but still had headaches (these continued for months). He was advised not to dive again.

The discharge diagnosis was marked constitutional DCI, with psychosexual dysfunction and short term memory problems.

However he was unable to carry out his occupation, as he was unable to remember what stage he was up to when working on vehicle repairs. He lost self confidence and came under financial pressure. He had a subtle deficit, not visible to others, which was impacting on job performance. A poor prognosis was predicted. He was treated with simple analgesics.

May 1993 Follow up

He had poor balance, appearing to be drunk and staggering with minimal alcohol. His SRT was 3 seconds and he was unable to stand on either leg with his eyes closed. He also had earache (left worse than right) with constant occipital headaches and tinnitus. Memory and cognitive deficits were present. He was depressed.

August 1993 Follow up

His SRT was now normal (30 seconds). His main problems were concentration and short term memory. There wa numbness of the sole of the left foot, and especially heel which ached. His right groin had ached for 3 months but there was no hernia or mass.

September 1993 Follow up

ENT assessment: Mild high tone sensorineural deafness, consistent with noise exposure. Hearing loss and tinnitus worse especially with low background noise. His spouse complained about loud TV. Previous history of noise exposure, shooting (none for 6 years) and occupational as a mechanic.

November 1993 Follow up

The neurologist assessed him as having no neurologic abnormality (!) but depressed. Recommended referral to a psychiatrist.

February 1994 Follow up

Headaches, tinnitus, and memory problems continued. He made silly mistakes at work. Still depressed. Unsure of bladder volume when passing urine. Work output about 60%. Symptoms of peptic ulcer.

January 1995 Follow up

Neurologically normal on examination. Good balance and gait.

July 1996

He was scrubbing the bottom of a boat on snorkel became tired and completed the job using scuba. (2 dives in 3 years.) After 30 minutes at 2 m (6 ft) he dropped the scraper and descended to 6 m (20 ft) briefly to recover it. Ten days later he had an itchy back but his memory was fine.

September 1996

He came to see me for a second opinion. For years he had had pins and needles in both feet, left more than right. He also complained of a burning pain with

defecation. He had a similar pain in both ankles and heels, which were both quite numb with reduced pinprick sensation. He had a prickly sensation in his right eyebrow, both eyelids and inside his mouth. He was taking fluoxetine hydrochloride (Prozac) at night and naproxen. A trial of clonazepam (Rivotril), 0.5 mg mane, was effective within minutes of administration. On this he slept better, the pins and needles virtually went and he was happy for the first time in years.

December 1996

The Accident Compensation Commission refused to authorise any further treatment for DCI. He had constant pain in both shins, hands and feet. Feet numbness had returned. But clonazepam (Rivotril) was effective for skin itchiness. He had variable skin sensation, reduced pin prick anywhere. He still was often dropping tools, forgetting where they were. He had poor libido (marriage fine) but normal erections. Lethargy was constant but his memory was improving.

February 1997

He was still very fatigued. After 2-3 days at work he had to go home to rest. His mood was stable but he had decided to sell the business. His alcohol intake was minimal. He was on fluoxetine hydrochloride (Prozac) 20 mg nocte, clonazepam (Rivotril) 0.5 mg mane and naproxen prn, which had helped. On examination his SRT was 30 seconds and he had a shorter gait step.

April 1997

Hands were seizing up. He had to straighten his fingers out with the other hand. Dropping tools was very frequently through inattention only, a daily hassle. Naproxen was helpful, but he was fed up and the business still for sale. He could not continue with his job.

In short a poor outcome.

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## OXYGEN THERAPY EQUIPMENT A THEORETICAL REVIEW

Michael Davis

**Key Words**

Accidents, equipment, first aid, oxygen.

**Summary**

The basic needs for oxygen therapy equipment are control of inspired oxygen concentration, prevention of carbon dioxide accumulation, minimal resistance to breathing, efficient and economic oxygen use, adaptability to different gas mixtures and adaptability to different modes of respiration. Understanding the performance characteristics of oxygen therapy devices enables better selection of equipment for diving accident management. Physiological studies have shown that these devices may be subdivided into *fixed performance* and *variable performance* systems. The *fixed performance* devices, when used properly, supply the predetermined oxygen concentration irrespective of the patient's ventilation characteristics. *Variable performance* devices provide variable oxygen enrichment (always less than 100%) depending on the interrelationship of oxygen flow, device factors such as functional apparatus dead space and patient factors such as the peak inspiratory flow rate. For supporting diving operations, ruggedness of construction, simplicity of design and use, ease of training and maintenance and purchase price are all of importance. The newer demand regulator and rebreather systems (both fixed performance) in robust casings are well suited to the early care of diving accidents. However, they are moderately expensive, may require considerable training and carry an obligation on the part of the user to learn, and maintain, airway management skills.

### Introduction

Oxygen therapy is an important component of the early management of many medical and trauma emergencies including diving accidents. All ambulance and field medical rescue teams carry oxygen as an integral part of their equipment and there are virtually no emergency situations in medical practice in which oxygen could potentially be harmful administered in high concentrations for short periods.

Unfortunately, medical students have been taught for many years about the potential dangers of oxygen therapy in a small group of patients with chronic obstructive pulmonary disease who are dependent on an hypoxic drive for their continued spontaneous respiratory effort. This view has now been largely discredited.[1] In addition, the pulmonary toxic effects of high concentrations of oxygen,