## LATE SEQUELAE OF DECOMPRESSION SICKNESS
### A case report

Allan Sutherland

This is the story of T, a 41-year-old male diver who has suffered, and continues to suffer, significant psychological and physical disabilities after two episodes of decompression sickness in July 1987 and February 1988. The medical assessment of this man has been difficult, with many reports conflicting, resulting in delays with compensation.

He suffered his first diving accident in July 1987 during his second dive of the day. This dive was to greater than 30 m looking for crayfish. He became wedged under a rock and was only able to free himself by removing his backpack buoyancy compensator and using his octopus regulator. Separated from his gear, but holding it, he surfaced rapidly.

By the US Navy tables he exceeded the no-stops dive limit by at least 19 minutes. He was therefore a candidate for both decompression sickness, by virtue of the length of his dive, and air embolus, because of his rapid ascent.

On the surface he felt "funny" and noticed tingling of his lips, fingers and toes. Subsequently, 3 hours later, he developed pain in his left shoulder and right hip. He was unable to pass urine and had an unsteady gait.

On examination, both the left shoulder and right hip were painful on all movements, with associated weakness around each of these joints. He had hyperaesthesia of his left arm and loss of sensation to pin prick over the dorsum of his right foot.

He was treated with an extended US Navy table 6, receiving a total of 12 hyperbaric oxygen treatments.

Two and a half weeks after this accident a neurologist reported that T had a slight pyramidal weakness on the right side and extensor plantar responses on that side, with pin prick and position sense impaired in the right leg. He was unsteady standing on either foot, even with his eyes open. The neurologist's report included "I agree that this man has had decompression sickness affecting the cerebral hemispheres and spinal cord. I would expect him to make a complete recovery from these problems within 3 months."

Contrary to medical advice, T resumed diving and sustained a further diving accident in February 1988. After a late night, and excess alcohol, T did a strenuous dive for about an hour at 18 m. He became muddled, and developed pains in the hips and surfaced. He saw flashes of light at about 5 m but he remembers very little until he was put in the recompression chamber.

T's worst symptom continues to be pain in the right hip. He has a balance problem and does not seem to empty his bladder properly. He claims that his concentration is not as good as it should be. He tends to drop things but has not noted any specific impairment of his hands.

On examination recently there was slowness in performing rapid alternating movements of his arms, with possibly some slight impairment of position sense. He was mildly ataxic, walking with his feet a few inches apart. There was impairment of position sense bilaterally, with hyperaesthesia in the right leg. The tendon reflexes were reduced and the plantar responses were flexor.

Now the neurologist comments "This episode of bends seems to have produced mainly spinal cord problems which are relative minor and should recover completely. From a neurological point of view, I consider that he will, again, make a complete recovery".

There are now, in all, 16 reports available on T. Many are conflicting in their content and clearly demonstrate the great difficulty the medical profession has in appropriately assessing the long-term effects of decompression sickness, especially the personality changes and soft neurological deficits.

In an attempt to help this man two neuro-psychological assessments were done. The first was performed in November 1988 and, nineteen months later, the second in July 1990. The test performed were Block Design, NART, PASAT, Picture Completion (WAIS-R), Quick Test, Key Complex Figure, Stroop Test, Trail Making Test, Visual Sequential Memory, Visual Reaction Times (two forms), Wechsler Memory Scale (Form II) and Word Fluency.

The final neuro-psychological report states that T was having a lot of difficulty concentrating and was unable to cope with any situation where things were happening fast, including situations were there was a lot of noise, where several people were talking at once. This difficulty affects his family and social relationships and has implications for his ability to work. When his children are noisy, or racing around, he gets very irritated and cannot handle it. Similarly, in many social situations, he is unable to follow conversations and just switches off. Consequently there are many social contacts that he now avoids.

When he wants to write "It all gets jumbled up". Although he is able to drive a car, he does not do this often as he has to put so much effort into it and after driving feels exhausted. Both there difficulties are consistent with impaired ability to concentrate and a slowing of the thinking

process.

Difficulties of a more physical nature included difficulty sleeping more than a few hours a night although he feels worn out, difficulties with sex, which include variable difficulty in gaining an erection, also pain and lack of feeling during intercourse, and a reduced tolerance to alcohol.

He is learning to accept his intense frustration and anger, adapting to frustrations by avoiding them. T feels anti-depressants have been helpful, not just to improve the mood state, but they also permit him to tolerate his disability and its frustrations. However his wife thought they made him more aggressive.

This man has been left with the kind of cognitive impairments that frequently follow other forms of diffuse brain damage. The pattern of his neuro-psychological profile is strongly suggestive that he has organic dysfunction of the brain. This continues to have a profound impact on his family relationships and his ability to work.

**Discussion**

After some 3 years, and many conflicting reports, T is still awaiting final permanent disability settlement payments. He continues to have a poor attention span, impaired concentration, reduced short-term memory, depressed mood, labile emotions, language difficulties, impaired balance, weakness of his left shoulder muscles, occasional paraesthesiae in the right leg, episodic diarrhoea and urinary hesitancy.

Despite these crippling problems we have optimistic neurological reports in conflict with the neuro-psychological assessments which are more in keeping with our diving medical assessments. Others have noted that "The cerebral recovery from DCS and AGE is more refractory than previously thought".[1] T is not alone, he is one of 8 divers who were unable to return to their previous employment, because of persisting disorders of their higher intellectual function and mood state, after hyperbaric treatment at HMNZS PHILOMEL for a diving accident.[2] There were 30 patients in the series reported. These 8 patients were assessed as impaired using the criterion of return to usual employment. The patients were invited to make self-assessments. Assessment was also made by the spouse and by the family practitioner. In addition psychometric testing was performed at the Post-Concussion Clinic, Auckland Hospital.

All these assessments gave evidence of disturbed higher intellectual function and mood state, with considerable variability of the symptoms. Although the assessments have a large subjective component, and the report is brief, the conclusion is statistically significant (8/30) and the patients' disabilities are very real. The paper's conclusion was "That the cerebral effects of sport diving accident cases are refractory when assessed by family observation, G.P. observation, and psychometric testing".

This case clearly demonstrates the difficulties in quantifying the late sequelae of decompression sickness and achieving appropriate compensation payments.

**References**

1 Curley MD, Schwartz HJC and Zwingelberg KM. Neuropsychologic assessment of cerebral decompression sickness and gas embolism. *Undersea Biomed Res* 1988; 15 (3): 223-236

2 Sutherland A. Diving accident cases treated at HMNZS PHILOMEL recompression chamber in 1988. *SPUMS J* 1990; 20 (1): 4-5

*Dr Allan Sutherland, MB, ChB, Dip DHM, is a founding member of the Diving Medicine and Assessment Centre (DMAC), 4 Dodson Avenue, Milford, Auckland 10, New Zealand.*

## FREMANTLE HOSPITAL HYPERBARIC MEDICINE UNIT THE FIRST YEAR

Harry Oxer and David Davies

**Introduction**

After 12 years planning, and annual submissions to the Government, the Hyperbaric Unit at Fremantle Hospital was opened for business on November 27th, 1989. Construction was directed by Hyox of Scotland but most of the work was subcontracted to local West Australian companies.

The Unit is located in a former laundry and has facilities for consultation and wound care in addition to the administrative and treatment areas. Its effectiveness was justified by achieving the planned operational goals within six weeks rather than the expected six months.

The treatment unit consists of two hyperbaric chambers connected to a separate entrance lock which has provision to lock-on a transfer under-pressure module (Figure 1).

"Mildred", as the main treatment chamber is known, is a vertical cylinder with an internal diameter of 3 m and is 3 m high. It has a large rectangular door through which patients can walk or be wheeled on trolleys. The door is large enough to allow a Drager Duocom transportable chamber to