SPUMS ANNUAL SCIENTIFIC MEETING 1999

DECOMPRESSION ILLNESS IN THE ROYAL AUSTRALIAN NAVY 1961-1999

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Key Words
Decompression illness, history, treatment, sequelae.

The 1960s

In 1961 the Royal Australian Navy (RAN) determined it had a requirement for an underwater medicine service. The Underwater Medicine Clinic was opened in February 1961 at HMAS RUSHCUTTER, a shore establishment where the divers were based. The RAN coerced an anaesthetist, Rex Gray, to accept a commission in the Navy and appointed him to the job. The requirement for an underwater medicine clinic was confirmed on his first day on the job when a diver conducting free ascent training suffered pulmonary barotrauma and was dead on arrival at the clinic. That was Rex Gray's introduction to underwater medicine.

The RAN further recognised there was a need for an underwater medicine research function, and laboratory facilities and civilian technical staff were provided. The School of Underwater Medicine (SUM) was officially established on 21 January 1963. SUM was relocated to HMAS PENGUIN from RUSHCUTTER in July 1968. Going back through the records, there are abundant press clippings relating the involvement of the RAN in making dramatic dashes across the country to rescue divers in distress. At that time, the RAN provided the major treatment facility for injured divers.

When SUM moved to HMAS PENGUIN, "the offices, library and laboratory were installed in a part of an old, ramshackle wooden building that is 200-300 yards away from the diving working area. The floors were bare, with gaping cracks, the walls and fibro roof were uninsulated and out of alignment." SUM remains in a similar building and no major structural modifications have occurred over the last 30 plus years!

In 1968, the Navy was still averaging one diving death a year. On average, one diver a fortnight was reported as losing consciousness during a dive on a rebreather set. Originally, diver error was considered the most likely cause of these incidents, until Carl Edmonds and his staff studied the equipment. Volunteers, including Carl, exercised to unconsciousness in the water while medical staff hovered above, retrieving the unconscious diver and collecting gas and blood gas samples for analysis. The answer was hypercarbia due to inadequate carbon dioxide absorbent systems. It would be difficult to obtain ethics committee approval for that type of experiment today!

The 1970s

It appears the first widely published work from the unit on decompression sickness was in 1976. Both the Singapore and Australian navies were interested in the types of diving accidents that were occurring. As it still is today, the distinction between Navy and civilian or recreational divers, was very marked. Navy divers had a disproportionate dominance of joint bends compared to recreational divers in whom cardiorespiratory and neurological symptoms were far more frequent. Jimmy How, Dawn West and Carl Edmonds reviewed a clinical series of 115 cases of DCS in civilian divers and, even at that time, commented that the classification between Type 1 and Type 2 decompression sickness (DCS) was not wholly satisfactory.[1] The Singapore Armed Forces recompression chamber treated 40 divers, while SUM treated 75. To be part of the study, the diver had to have had indisputable signs of decompression sickness or development of clinical symptoms during or after ascent, which were relieved or cured by recompression. They did not include cases of pulmonary barotrauma.

Various treatment tables were used. The air tables were still used widely, particularly in Singapore as well as the 18 m oxygen tables. However, Carl Edmonds liked to use what he called his "high oxygen pressure" tables, which was the maximum safe oxygen pressure, administered either in an RCC or underwater. He described this treatment as going to depth of relief on an appropriate PO_2 and staying on oxygen for the ascent. He did not give air breaks and followed up the treatment with 100% oxygen after surfacing. Basically these tables involved recompression to 9 or 12 m on 100% oxygen for periods of 2-3 hours. Carl believed there was a lesser risk of oxygen toxicity compared with the 18 m tables and he used a much slower rate of ascent, 12 minutes per metre. He did however qualify the use of his tables and he is quoted as saying, "You should only use these tables if you are a prudent diving physician who can predict outcome and final prognosis from the dive profile, the time sequence of events, and the clinical presentation. Non-experts should adhere to the strict guidelines detailed in the shallow oxygen tables."

The patients also received adjunctive treatment according to the severity of their presentation. Some received intravenous fluids, catheterisation was performed as necessary and a number of patients received steroids. Anti-epileptics and tranquillisers were administered to

neurological cases and electro-diagnostic and clinical monitoring was used as required.

Although all the divers were civilians, they ranged from recreational divers to abalone divers and locally employed divers, using either scuba or surface supply. The mean age was 32.4 years. The mean depth dived was 30 m and the mean duration of the dives was 120 minutes. The mean time of onset of symptoms was 33.1 minutes from the time of leaving the bottom. Eighty-seven (76%) exceeded any dive tables which were available. For 13 (11%) there was not enough data to say whether they were within the tables. There were only 15 (13%) who had dived in accordance with tables. The mean delay to definitive treatment was 50.9 hours.

Fifty-four divers (47%) presented with what was then known as Type 1 decompression sickness or joint pains. Upper limbs were the most affected, shoulders and elbows, followed by knees and hips. When multiple joints were affected, they tended to occur in neighbouring joints.

Sixty-one divers (53%) presented with Type 2 DCS namely cerebral, spinal, or combined cerebro-spinal, inner ear and cardio-respiratory disease. Spinal lesions tended to predominate in the cases from Singapore, where there were many fishermen divers with long deep dives, whereas in Australia, cerebral and cardio-respiratory symptoms seemed to be more common.

Many of these cases of Type 2 DCS had initially been treated with in-water air recompression. This was often aborted because of the difficulties encountered and the patients often surfaced far worse than at the commencement of the treatment. This procedure also often resulted in an unnecessary delay to definitive treatment. Spinal cord involvement was the most likely cause of persistent disability at that time.

Fifty-seven percent of patients (66) had symptoms within 10 minutes of completing their dive. Twelve divers (10%) developed symptoms during repetitive dives, on ascent or while decompressing. Thirty one (27%) developed symptoms immediately on surfacing and twenty three (20%) within the first 10 minutes. The longest duration between ascent and initial development of symptoms was 19 hours.

How, West and Edmonds used a simple grading system to assess the response to treatment. A complete cure was assessed as a grade 4 response; death was assessed as a grade 0 response, with partial responses in between. Ninety five divers (83%) had a complete or almost complete cure (grades 3 and 4). They did well with whatever treatment table was used. This included a mixture of air tables, shallow oxygen tables, surface oxygen, or Carl's high-pressure oxygen table. Two divers died and 14% (16 divers) showed no improvement.

Reviewing this sort of treatment is difficult because the severity and local conditions decided what treatment table was used. Mild Type 1 decompression sickness occurring in a patient remote from a recompression chamber, e.g. in Vanuatu, was likely to have been treated with 100% oxygen at surface pressure. In Singapore, they tended to use air tables, while in Australia, Carl Edmonds tended to use his own oxygen table. Looking at the graded responses to treatment, Carl's high-pressure oxygen table was assessed as having the best response and surface oxygen achieved better results than the deep air tables.

In the majority of cases, surface oxygen was administered, following completion of the recompression, for an hour on, an hour off, for up to 24 hours. A lot of the divers, even at that time, but particularly for spinal decompression sickness, were given a follow up hyperbaric treatment. The initial results indicated 60% (69 divers) achieved full recovery within 24 hours of their first treatment. A further 17% (20 divers) recovered within one month and 14% (16 divers) within one year. Only 7% (8 divers) were permanently affected and two divers (2%) died.

The authors compared the use of the shallow oxygen tables against the air tables. They thought that, certainly in military diving, the shallow oxygen tables had a dramatic effect and were seen to be the treatment of choice. This was not so for the civilian divers, because they were diving outside the tables, had more significant disease and presented later compared with the military cases. Certainly, the lack of any sort of decompression staging, inadequate equipment and equipment failure at depth contributed to the severity of the disease in the civilian divers and the lack of facilities for decompression played a role in the delays to treatment.

How et al. agreed that the shallow oxygen tables (US Tables 5 and 6) were much more attractive to the operators. Using them meant the staff did not have to spend days at the chamber as they did with the saturation air tables. They also felt that in-water recompression worked in remote areas when performed properly. Carl was certainly one of the main proponents of in-water oxygen therapy. Safe in-water oxygen therapy requires an adequate supply of oxygen, a full-face mask and a platform for the patient. The patient must be kept warm and be under the continuous observation of an underwater tender. Sheltered water is preferable.

The 1980s

The 1980s saw a change of personnel at SUM and can be described as the Des Gorman era. There was beginning to be some doubt as to whether the US Tables 5 and 6 were adequate. Some patients, particularly the paraplegics and the serious neurological presentations, did not seem to respond. The philosophy at that time at SUM

was to not surface the patient until there was full resolution of all symptoms. So they would start off with a Table 6 and, if there was no response, go to 30 m, then to 50 m and then proceed to an air saturation table. There are no published reviews of the treatment at that time, but after about 3-4 years of following this treatment philosophy, the return on that investment of time, staff and improvement in the patient was considered very poor. The desire to do air saturation tables soon passed. Instead, the philosophy changed to include a RN Table 54. This is a long air table, of approximately 39 hours. But so few experienced significant recovery with this table, that SUM changed to a short stay at 50 m, followed a RN Table 63, with a total time of only about five and a half hours.

At this time, SUM started to look at outcomes from treatment and neurological sequelae.[2] This review period went from 1984 to 1986. They studied 88 divers, one of whom had an air embolism and the rest were assessed as having had decompression sickness. All were treated in the RAN multi-place chamber and all were intravenously hydrated. Initially, they were treated at 2.8 bar on 100% oxygen (USN Table 6). If there was no response they were changed to an oxygen-nitrogen mixture and compressed to either 30 m or 50 m. Recurrent or persistent symptoms or signs were treated with daily hyperbaric oxygen (HBO). HBO was given at 9 m, 10m or 14 m for 2 hours, depending on who was running the treatment. They examined the patients clinically for neurological outcome at one week, one month and one year. The patients underwent a formal psychological assessment. The patients had 19 lead EEGs under conditions of rest, with photic stimulation and with hyperventilation, and underwent a CT head scan. When the EEGs and the CT head scans were done either a neurologist or a radiologist who was not aware of the patient's history evaluated the patients.

Before any treatment, a neurological deficit was detected in 68 of the 87 patients (78%) with DCS. At discharge 84 patients (96%) said that they were completely asymptomatic. Clinically no abnormality was detected. Twenty out of 61 (33%) had an abnormal psychological assessment on a battery of profiles, 22 (36%) were assessed as having abnormal EEGs and 8 (13%) had abnormal CT head scans. At follow up the numbers dropped off at a week, a month, and a year. After one week only 46 people (or half) presented for review. At discharge 96% were said to be completely normal, but a week later ten people (22% of those who attended) were detected, clinically, to have a neurological deficit. After one month, the numbers dropped again, but there was some recovery. While 10 were considered abnormal clinically at one week, by one month only 2 of those had persistent clinical signs.

Diver follow up is a problem with all our diving studies. There is a high dropout rate. The important thing was that the clinical morbidity at discharge was considerably lower than when the patients were followed up a week later. We may believe that we have good recovery at discharge, but if we do not follow up our patients we are not sure what is happening to them. After this work, there was certainly belief, in Australia and perhaps worldwide, that USN Tables 5 and 6 were not always effective.

Michael Loxton[3] undertook a study to determine the effectiveness of the Minimal Recompression Oxygen Tables, with the aim of trying to provide an accurate estimate of the failure rate and looking for predictors of poor outcome. There were 319 patients treated between January 1983 and December 1993. Fifty-six patients were excluded: insufficient information was recorded in 12 cases, some were later assessed as not having DCS and other cases had treatment on air or mixed gas tables. 263 patients were finally included in the study.

The mean age of the divers was 29. At that time, it was probably representative of the wider diving population that 83% (218) of the study group were male and 17% (45) were female. Most were treated on a USN Table 6, some with extensions. Others had a Table 5, and some were given a 9 m oxygen soak. A mean of 2.2 additional hyperbaric treatments was administered.

At the end of the first treatment, 153 divers (58%) still had residual symptoms. At the end of all treatments 76 divers (29%) still had residua. There continued to be an improvement, from one week to one month, to six months, to 12 months post treatment. However, even at 12 months, 24 divers (9%) had residual symptoms.

Patients who presented with neurological decompression illness were more likely to fail to respond fully. There was a significant number who had residual symptoms at the completion of all treatments. Thirty four percent of the neurological patients had residual symptoms, compared with 21% of the non-neurological, or the old Type 1, patients. This was a statistically significant change. When subdivided into sports divers, (who were the majority), naval divers, and professional divers the differences in poor outcome were not statistically different between the three groups. The proportion of Naval divers with Type 1 symptoms was higher, but not statistically different.

When looking at outcomes, Michael Loxton looked for associations between 17 variables and the presence of residual symptoms at the completion of all hyperbaric treatments. Seven variables were associated with a statistically significant increased risk of treatment failure. Those were:

the presence of neurological disease;
age over 35;
going to altitude after the dive;
failure to comply with DCIEM tables;
further diving after onset of symptoms;

treatment delay;
lack of first aid oxygen.

However when adjusted for confounders, the only variables that had significance with poor outcome were age over 35, treatment delay and further diving after onset of symptoms.

Seventy-six (29%) of 263 patients were left with residual symptoms. The shallow oxygen tables appeared to be more effective for non-neurological than for neurological DCS and neurological symptoms appeared to be more likely to persist after completion of all treatment. Certainly, he demonstrated a tendency for the residual symptoms to improve spontaneously. One of the conclusions was that the shallow oxygen tables appear adequate for the old Type 1, or non-neurological, symptoms but perhaps we should be seeking different treatments for neurological disease.

The 1990s

I believe that there are different subsets of patients. We see the patients who come in a week after diving with some mild paraesthesia and they just do not feel right. I treat them with Table 6 or an extended Table 6 and it seems to work quite well for them. Another group is patients who come in with serious, progressive, neurological disease and who, despite early surface oxygen, despite early recompression and despite aggressive fluids, do not seem to get better. That produces difficulty for us at the clinical level. The 22 year old who leaves the chamber paraplegic is a treatment failure and a significant burden for the community. My treatment philosophy is that the very acute patients, with progressive disease, who present within 24 hours of their injury and are not resolving at 18 m, are recompressed to 30 m. I use 50:50 heliox at that depth. Why? Why not? We do not know whether it is the right thing to do, but that is what I have elected to do. In the last 5 years, we have had a number of patients who have not done well at 18 m and who have resolved at 30 m. We have also used lignocaine for some of those.

There are divers who dive on open circuit, using trimix (helium, nitrogen and oxygen), to 78 m off Sydney. There was a group of 5 such divers, two of whom have died as a result of this activity. Two of the survivors presented a week apart after one of these deep dives. The first presented with shoulder pain and weakness in his arm. He deteriorated at 18 m, so I took him to 30 m on heliox. He responded very well and was asymptomatic after that treatment and at follow up. His buddy, who presented a week later after diving a very similar profile, had shoulder pain, arm pain, weakness and paraesthesia and was treated by one of our other physicians on an extended USN Table 6. He was very slow to respond and had to have a number of follow up treatments. He was still symptomatic at discharge. This is not statistical evidence, but anecdotally, it influences our decisions.

The Next Millennium

I am not sure what treatments we will be using, however we can only await the outcome of a double-blind randomised multi-centre trial assessing treatment outcome, to help guide our clinical decisions.

References

1 How JH, West D and Edmonds C. Decompression sickness in diving. *Singapore Medical Journal* 1976; 17 (2): 92-97

2 Gorman DF, Edmonds CW, Parsons DW, Beran RG, Anderson TA, Green RD, Loxton MJ and Dillon TA. Neurologic sequelae of decompression sickness: a clinical report. In *Proceedings of the Ninth International Symposium on Underwater and Hyperbaric Physiology*. Bove AA, Bachrach AJ and Greenbaum LLJr. Eds. Bethesda, Maryland: UHMS. 1987: 993-998

3 Loxton MJ. *The outcome of 263 divers with DCI treated with minimum recompression oxygen therapy*. Master of Public Health Thesis: University of Sydney, 1996.

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TEN YEARS OF TREATING DIVERS

Chris Acott

Key Words
Accidents, asthma, decompression illness, flying after diving, incidents, sequelae, treatment.

Introduction

I am going to discuss of some of the clinical gems that I have gathered over the past 10 years. I think it was Carl Edmonds who said “How do you know when a diver is lying to you? You just have to watch his lips move.” That