antiocoagulated, sometimes they get steroids and they are fully oxygenated. What a perfect set up to be taken to a chamber to be fixed and one would think the survival should be excellent.

Unfortunately one of my first experiences was a 39 year old woman operated on for an atrial septal defect. She got some air because of the technique and they did not do anything about it, they just basically let her go. It is only recently that a chamber has been put in that city. That represents the sort of problem that cardiac surgery can present. There is one surgeon in the US who operates in the chamber. He has been jumping up and down for years and no one has ever listened to him. Cardiothoracic surgeons are not the only ones in medicine who create that sort of a problem. People on dialysis have the problem with their arteriovenous fistulae when the dialysis machine infuses air into the patient. Neurosurgery also does the same thing to people sitting up when, especially in the posterior fossa approach, air can be sucked into a vein because of the low venous pressure. These require the same sort of recompression treatment.

Dr A Slark

I have a suspicion that this sort of case presents great concern to the people managing it because the physician is alarmed at the sight of blood and the surgeon always wants to do something, actively to put his finger on the leak. As soon as you put a patient in a chamber you have taken him away from the person who can actively stop the bleeding. Did you have a great deal of discussion about this particular pattern of management in this most unusual case?

Dr A.Santos

No. The uniqueness of the situation dictated that he just go to the chamber and no one thought any more about it. It is only one case so one can either postulate nothing or conversely one can postulate everything. It is certainly a bad time. One needs to have somebody in there with him, one needs to maintain the airway in the chamber and if something does go awry or persists too long one needs to do something about it. With diffuse pulmonary haemorrhage that would be very tough problem. Conversely if it was bronchial artery rupture we now have the capability of putting a catheter into the site of the rupture and embolising gel foam or clots into the artery and stopping the bleeding that way.

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HYPERBARIC MEDICINE IN CHRISTCHURCH, NEW ZEALAND, 1979-88

Michael Davis

Introduction

In 1980 at the SPUMS meeting in Singapore, I reported on the establishment of a new hyperbaric chamber at The Princess Margaret Hospital (TPMH), Christchurch, and our first year of activities[1]. The chamber was then, and still is, the only hospital-based multi-place chamber in New Zealand. Monoplace chambers exist in hospitals Dunedin (currently non-operational), Auckland and Wellington (operational). Until a decade ago, the only chamber actively treating divers was that at the Naval Base in Auckland, and even today about 80% of all decompression sickness (DCS) and cerebral arterial gas embolism (CAGE) cases in New Zealand are treated there.

The Christchurch facility's history is an interesting one. In brief, the chamber and a small compressor were donated to the Hospital Board by the local diving community in 1979 following a vigorous fund-raising effort. The whole thing was installed on the cheap, the Board undertaking to house and maintain the unit, but no funds were earmarked to establish a clinical service, staffing was to be on a voluntary basis.

The chamber was generally regarded to be a White Elephant, and Harry Guy (a respiratory physiologist now working for NASA) and I to be a pair of eccentric quacks. However, the referral of 33 patients in the first 15 months brought about something of a crisis, forcing the Hospital Board to address rather more constructively the provision of a "service", however rudimentary. Board policy was and still is that we may provide an acute emergency service only, and that there would be no additional staffing establishment to do it! Nevertheless, within these limits the Board has been supportive in many small ways over the years to maintaining our rudimentary service. At a time of severe financial constraints in health care in New Zealand, the establishment of formal hyperbaric medical services is likely to remain a low priority compared to many other perceived needs.

Despite the voluntary nature of the service, we are surprisingly well staffed. Medical cover is currently provided by 7 doctors, 3 consultants (2 anaesthetists and a geriatrician), 3 registrars and a family physician, all on a ring-round basis, there being no obligation to make oneself available. Only the family physician is paid on a fee-for-service basis by the Hospital Board, the rest of us do it for free, over and above our usual commitments. Only I had previous hyperbaric or diving medical experience.

We have 10 chamber operators. Four are Board employees, whilst the other six are local divers on whom

we rely heavily. Patient attendants are drawn from a voluntary pool of TPMH nurses who have undergone an introductory 18 hour hyperbaric nursing course run by me. Nurses are generally called when on duty. They are free to decline the call if they wish to. We try to keep our pool above eight to ensure nursing availability on this emergency basis. As you will appreciate, these arrangements do not make for an efficient service, yet the whole thing works surprisingly well because of the enthusiasm of all those involved. In fact, we have a waiting list of people wishing to work in the chamber!

Monoplace Chamber, 1972-78

Christchurch was no stranger to hyperbaric medicine even before 1979 as a Vickers monoplace chamber was used from 1972 for a trial of hyperbaric oxygen (HBO) in head and neck cancer. Between 1972 and 1978, 108 patients, 87 in the cancer trial, 18 with presumed clostridial infections and 3 others received a total of 693 HBO sessions. Apart from minor aural barotrauma in some patients, the only complications of therapy were three oxygen convulsions, none of which resulted in any morbidity. The Vickers chamber was sold to the Royal Adelaide Hospital in 1980.

Clinical Experience

Our clinical experience in the last nine years is summarised in Table 1. What lessons have we learned? Whilst this is a small case load by world-wide standards it has certainly been a learning experience for all those involved.

CLOSTRIDIAL INFECTIONS

Our Christchurch experience in the management of Clostridial infections since 1972 was reviewed two years ago[2]. There is little to add since then, except to note that over the last five years there has been a definite fall in the number of cases referred. This seems to reflect a real reduction in the incidence of this devastating infection, perhaps related to changing practice in the use of prophylactic antibiotics for surgery and trauma.

CEREBRAL ARTERIAL GAS EMBOLISM (CAGE)

Our overall experience with CAGE is too limited to draw useful conclusions from, except to say that we have not been vigorous enough in the use of HBO for this condition. Other disease entities such as osteoradionecrosis represent only a smattering of cases. I therefore propose to review only our experiences with decompression sickness (DCS) and carbon monoxide (CO) poisoning.

DECOMPRESSION SICKNESS

Of the 49 cases with a presenting diagnosis of DCS, six were considered to have musculo-skeletal problems unrelated to diving, whilst seven patients were managed, some in the monoplace at Wellington Hospital, by the Critical Care team in phone consultation with ourselves, leaving 36 patients who were managed in Christchurch.

Six of these patients were not given hyperbaric therapy: One refused treatment, three were observed in hospital and two late (over 10 days) referrals were declined treatment as no longer acute emergency cases. Reviewing the medical records of the latter five patients, the decision not to provide hyperbaric care was incorrect in all, and follow-up was incomplete, so whether they made a full recovery is unknown. Clearly, in suspected DCS, observation is not sufficient, hyperbaric oxygen therapy is required. A patient whom we initially managed incorrectly exemplifies this basic rule:

TABLE 1
CHRISTCHURCH PATIENT REFERRALS, OCTOBER 1979 - DECEMBER 1988

Presenting Diagnosis	Referred	Treated
Decompression sickness	49	30
Cerebral arterial gas embolism	10	3
Carbon monoxide poisoning	77	61
Clostridial infection	26	21
Radionecrosis (soft tissue and bone)	9	5
Miscellaneous	11	4
Total	**182**	**124**

Case 1

This 40 year old man undertook six dives (three on each day) to varying depths between 24 and 45 m (80 to 150 ft). His dive pattern grossly exceeded the no-stop limits but he undertook no decompression stops. He presented two days later to Wellington Hospital with mild persistent shoulder pain. It was decided by phone to manage him conservatively with anti-platelet therapy, high flow oxygen and observation rather than undertake the considerable expense of an air evacuation at that stage for mild symptoms.

By the following morning, he had developed neurological signs of a cervical cord lesion! He had hyporeflexia on the left side and areflexia on the right with bilateral up-going plantar responses. Power was diminished in the right arm particularly proximally. However, tone, gait and posture were all normal, with a negative Rhomberg's. There was diminished pin prick sensation over the right arm particularly the C5-7 dermatomes. He denied previous trouble with

his right arm. CXR showed a small area of atelectasis at the left base and a small amount of pleural fluid on the right.

Most of these features resolved with HBO but he remained slow mentally and hyporeflexic. With a history of over a decade of similar diving practice he presented very much the picture of the "punch-drunk" diver!

As our experience has grown we have become less impressed by any distinction between Type I and Type II DCS and their epidemiology. In our series only six patients had "classical" Type I symptoms alone, whereas 24 had neurological signs and/or symptoms. In nine, these features were combined and three patients also had respiratory symptoms at some stage. Thus in our experience, DCS in sports diving is a predominantly neurological disease, an impression seemingly shared by others. Whether the pain-only patients all console themselves in the pub is a moot point! Case 1 again illustrates the point that ALL DCS must be taken seriously. The costs of air evacuation of a diver with mild symptoms may seem awe-inspiring at the time but they fade into insignificance in comparison to the costs of neurological rehabilitation!

Another interesting aspect has been the chronology of symptom onset. Francis et al[3] have recently reviewed the onset of DCS in a large series of patients drawn from centres all over the world. Their data clearly demonstrates the acute nature of DCS with about 80% of cases having an onset time of *less than one hour*, 40% within 5 minutes of completion of the dive! In our small series the onset times were very similar to these reference data with 38% at 5 min, and 60% within one hour. By contrast, it took over 24 hours for two-thirds of the patients to reach our unit. Much of this delay was the fault of the patients themselves in failing to present for treatment for some considerable time. Two typical examples are:

Case 2

A 49 year old qualified diver with 9 years' experience, carried out a dive to 31.5 m (115 ft) for 20 minutes. He made too rapid an ascent because of bouyancy problems. About three hours later he developed chest tightness and felt uncoordinated in the right leg. Suspecting he had a bend, he went back down to 3 m (10 ft) for about 5 to 10 minutes, then breathed oxygen on the surface. This had no effect, but he went home anyway! Overnight he noticed continuing incoordination, weakness and paraesthesia in his right lower leg and difficulty passing urine. He finally presented to hospital the following day and was helicopter-evacuated to Christchurch. He gradually improved over several days of HBO, but has remained impotent since the accident.

Case 3

This 45 year old qualified diver with over 10 years' experience was wreck diving using an Orca "Skinny Dipper" dive computer. He did three dives. The first dive was 31.5 m (115 ft) for about 15 minutes. He then had a 2 hour 20 minute surface interval. The second dive was to 31.5 m (115 ft) for about 15 minutes. About 15 minutes after this second dive, during which he had a physically demanding swim with a heavy load of contraband, he noticed that he was unsteady standing and had pins and needles in his legs. He rested for a while and then elected to do a third dive to 21 m (70 ft) for 5 minutes with stops at 10 m (20 feet) for 5 minutes and 3 m (10 ft) for 10 minutes. That evening he developed progressive weakness and numbness from the waist down and had difficulty passing urine. However, he hid his symptoms from his diving buddy as they sheltered out overnight under a growing NW gale. The following morning evacuation was extremely difficult and he did not reach the chamber until mid-afternoon. During his evacuation a medical officer discontinued oxygen "because he was not cyanosed"!

On arrival, he was paraparetic to the T9/10 dermatome. Following 12 HBO treatments he had an incomplete recovery, but was able to return to work.

One message here is that the sports diving teaching agencies must teach new divers the importance of seeking out qualified medical advice early after a dive if they feel unwell. The modern trend with some agencies seems to emphasise the safety of the sport but to exclude any mention of such disastrous consequences as this to divers. Likewise, the medical profession needs to be better informed on diving medicine and its acute management, so that decisions such as that above concerning oxygen are not made!

Hyperbaric Management of DCS

It has been instructive to look critically at our management of DCS. In 14 patients, the initial treatment was a short oxygen table (US Navy Table 5), modified in five cases for the Wellington monoplace prior to transfer to Christchurch. One patient underwent a 10-hour unsupervised air treatment in a non-hospital chamber prior to referral. In the remaining 15 cases, a US Navy Table 6 was used, only three being with extensions.

The distribution of the number of treatments per patient is shown in Table 2.

In recent years we have learnt to treat more vigorously initially and provide repeat therapies more readily. Now Table 5 would be regarded by most as inadequate initial treatment and we now rarely employ it as the first treat-

Table 2

HYPERBARIC OXYGEN TREATMENTS FOR PATIENTS WITH DECOMPRESSION SICKNESS

Number of HBO	1	2	3	4	4+
Number of patients	15	7	1	2	3*

* these were given 5, 11 and 12 HBO treatments respectively.

ment except for 'missed decompression' in asymptomatic patients. In several cases the initial treatment was almost certainly too conservative and the patient was left with residual neurological signs at the end of his course of treatment. To be fair to ourselves, in the vast majority of patients a good outcome was eventually achieved, but the impression is certainly that we might have enhanced recovery rates by being a bit more vigorous initially. Because of our limited facilities and relative inexperience we have been reticent about embarking on very prolonged treatments. The following case illustrates this point:

Case 4

This 29 year old man undertook three dives in a morning, the first 30 m (100 ft) for 30 minutes, the second 42 m (140 ft) for 25 minutes and then 42 m (140 ft) for approximately 25 minutes. No decompression stops were undertaken. About 30 minutes after the third dive, he developed severe chest pain and painful joints. He then convulsed, remaining unconscious for about three quarters of an hour. During this time he was transferred to hospital and recompressed in a monoplace oxygen chamber to 3 ATA and held at this depth for 60 minutes before being decompressed and air evacuated by helicopter to Christchurch.

On arrival he was mentally alert and orientated to time and place but shocked, with a systolic BP of 70 mm Hg. He was completely paraplegic below T5/6. Some weakness of the left arm was noted, but this was not examined properly because a drip was in that arm.

After 40 minutes HBO at 60 ft when he had been given 2,000 ml lactated Ringer's solution, he was warm and well-perfused peripherally. He was now able to straight leg raise against moderate resistance, pin prick sensation was present, but still diminished from T7 on the right and T10 on the left. Light touch and proprioception were normal. Because of the considerable neurological improvement at this stage it was decided to treat him on an extended USN Table 6 with 2 additional 20 minute oxygen periods at 60 ft and 60 minutes at 30 ft.

However, when first re-examined after the ascent to 30 ft, there had been significant deterioration in power and sensation, and he was tachypnoeic. Despite this clinical deterioration, it was decided to complete the extended Table 6 as planned, finishing about 0400 hours. Despite 4,000 ml of intravenous fluids during HBO his haematocrit was over 0.65 at the end.

By 0800 hours he again had a complete T5 paraplegia, as well as diminished power in both arms, especially for fine hand movements, this being worse on the left than the right, and pins and needles in both hands. No objective improvement occurred with another Table 6, but by the end of two weeks of daily HBO, his arms had fully recovered, muscle power in the legs was grade 2-3, and he had deep pressure sensation in his buttocks and thighs. Three years later he is walking with the aid of 2 sticks but still unemployed.

The message is a well known one. Treat vigorously and to the maximum capability of your facilities. Treat repeatedly, until no further improvement is seen with each treatment, and do at least one more beyond that point.

Evoked Potentials Measurements

An area of assessment of DCS patients in which we have become interested recently is the measurement of evoked potentials (EP). The general view at the UHMS Hawaii meeting in 1989 seemed to be that somato-sensory evoked potentials (S-SEP) do not greatly contribute to management or research, since those with abnormalities invariably have clinical signs anyway. However, in addition to S-SEP, we have been evaluating brain stem auditory EP and visual EP. In several divers these have been abnormal without concomitant clinical neurological signs, and have shown recovery with time. We are beginning to collect some long-term follow-up data as well, but so far there is too little for further comment. It would be interesting to know whether other chambers have been using all three EP measurements on divers.

CARBON MONOXIDE (CO) POISONING

There remains disagreement in medical texts regarding the value of HBO in the management of CO poisoning. Despite this, in the author's opinion there is convincing laboratory and clinical data to support the view that HBO is the treatment of choice in CO poisoning. There may even be a case for urgent transfer of such patients from a distance, as recent clinical reports suggest that delayed HBO may also

be of benefit in preventing long-term sequelae. Readers are referred to a recent review in "Surveys of Anesthesiology" for an up-to-date statement on the evidence4.

Of the 77 patients referred for acute CO poisoning, 45 were from suicides and 16 accidental. Fifty seven were male, with ages ranging from 16 to 78 years. In the early 1980s it became apparent that a significant proportion of CO poisonings in Christchurch were not being referred for HBO. This was readily corrected by a medical grand rounds presentation, and the establishment of clear guidelines for referral. Since that time we have received 10 to 15 cases annually.

Our guidelines for referral are any of the following:

1 Asymptomatic with carboxyhaemoglobin (COHb) level of over 35% (now reduced to 30%);

2 A history of impaired consciousness at any time;

3 Neurological or behavioural signs and symptoms of any sort, excluding headache alone, on admission, irrespective of the COHb level.

Until recently the majority of patients had received only a single HBO treatment (either 60 minutes at 2.8 ATA or a US Navy Table 5). Increasingly we are now repeating HBO as our follow-up and liaison with the medical teams has improved. Recent evidence in the literature demonstrates that repeated exposures result in improved outcome.

The average admission COHb level was 33% (range: 2-57%). We place no reliance on the initial COHb in determining severity or prognosis. It is the clinical picture that is important. There was only a poor correlation between the admission COHb and the time interval between the end of exposure and sampling. Of the 77 patients, three died, and four were left with residual neurological problems. The % COHb levels were 11, 57, not known, 32, 39, 47 and 57 respectively in these patients. Thus, we have an overall treatment "failure" rate of about 9%.

It is widely believed amongst the general medical community that a CO poisoning victim suffering cardiac or respiratory arrest is unlikely to survive. In our series there were 8 patients who required Rescue Breathing or full CPR by ambulance teams at the scene of the exposure. Of these, four made a full neurological recovery, whilst two suffered residual neurological damage and two died. Clearly the outcome is worse in this group, but nevertheless the possibility of a successful outcome remains in all cases.

We were particularly interested to look at the delays in retrieval of these patients. Unfortunately accurate timing of discovery, ambulance retrieval, admission to the Emergency Room (ER) and commencement of HBO could only be ascertained in 33 patients from the metropolitan Christchurch area. In these 33 patients, the interval from discovery to HBO was two hours, ranging from one to five hours. We felt this was not particularly good for a city which you can drive across in under 20 minutes !

Of even greater concern was that most of this delay occurred in the Emergency Room, the mean time interval from arrival in the ER to commencing HBO being 96 minutes, with a range of 20 to 280 minutes. In a few cases this was the result of delays in finding one of us, but this was never more than about 30 minutes. Our task is clearly to engender a greater sense of urgency about HBO referral in junior ER doctors. In the last two years the St John Ambulance have been circumventing this whenever possible in serious cases by bringing victims directly to the chamber, rather than taking them through the ER. Looking at this problem chronologically, there has been a definite reduction in time delays in recent years compared to the early 1980s, which is encouraging.

The Future

So far the recompression chamber in Christchurch has survived on the enthusiasm of a small band of dedicated volunteers. However, this is not enough to ensure the lasting development of hyperbaric medicine in New Zealand, particularly when this field represents only a small portion of one's professional activities. Given the present crisis and dramatic changes in the delivery of hospital services in this country there is little chance of further developments in the foreseeable future being achieved from within our region.

However, several external pressures may change this. Firstly, there is an increasing awareness within the sports diving community of the need for properly funded hyperbaric facilities in New Zealand. In the last few years this has resulted in an increasing commitment from the New Zealand Underwater Association in this area, with the funding of the DES phone at HMNZS PHILOMEL, sponsoring of diving medicine courses and involvement in planning strategies for better hyperbaric facilities in Auckland. This is very much to be applauded, since without the public commitment of the diving community further developments are very unlikely.

Coupled with this, changes in the pattern of diving around our coasts continue to occur and may well force matters along. For instance, in South Island, in a little over two years the Mikhail Lermontov wreck in the Marlborough Sounds has claimed three lives and produced at least four DCS cases. This and the opening-up of Fiordland for sports diving, magnificent deep drop-offs in a geographically extremely remote region will surely present us with major problems in the years to come. In the last decade our diving accident referral rate in the South and Central regions has not risen, in sharp contrast to many other parts of the world.

For some years I have suspected this to be the calm before the storm!

What happens in the Auckland region will directly influence us in Christchurch. With nearly half the country's population centred there, there is little question that a major facility, similar in size to that in Adelaide, could be sustained and would be cost-effective. How the Minister of Health's Commissioner, Harold Titter, might be convinced of this, whilst looking to prune some NZ$ 60 million from the Auckland Area Health Board budget is an entirely different matter!

Finally, from the hyperbaric medicine viewpoint, developments in Australia are particularly important to us. It is essential that the new facilities being opened fulfil more than a service role, publishing high quality clinical and applied sciences research to back up the claims for hyperbaric medicine in various fields. Equally important will be the outcome of the UHMS submissions for the current Blue Cross/Blue Shield funding review of HBO. A successful defence of hyperbaric medicine in the United States over the coming year will be vital to the future development or otherwise of hyperbaric medicine throughout the world, let alone in Christchurch, New Zealand.

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THE BITE OF THE WAHOO

J McKee

The Wahoo is a fish of the tuna/mackerel family and resembles the Spanish Mackerel. It grows to about 45 kg (100 lb) in weight and six feet in length. It is very streamlined and is said to be the fastest fish in the ocean, at times reaching speeds of 80 km/hr (50 mph). It has a very large mouth and very sharp teeth with serrated edges and incredible cutting power with its jaws. The distribution of the Wahoo is mainly around Australia from the North West of Western Australia, across the northern coast, along Queensland and New South Wales as far as Lord Howe Island. The fish is a renowned game fish and Hemingway reports it is the best eating of all the game fish. The fish feeds by catching its prey and biting off the tail then devours the rest at its leisure. It is a fast game fish which is also good eating.

My interest arose by accident when asked to see a patient who had been bitten by a Wahoo. This was the first time I had seen this injury and I present it to reveal how incredibly sharp the teeth are. The teeth are one of the wonders of the sea. As far as I know they do not attack humans in the water. Usually the bite is an accidental attack on a fisherman or by some one brushing the skin against the Wahoo's teeth.

The patient was a Melbourne builder on holiday, who went out Marlin fishing and accidently caught a Wahoo. Just as it was brought into the boat, it decided to have its recognised last bite at anything close and it managed to seize the patient's lower leg. One single bite. When this solidly built builder presented to the Casualty Department he had no extensor power in his ankle, it was just a floppy foot. Under anaesthesia the reason for this became apparent. There were 2 wounds, a horizontal wound at the level of the medial malleolus about 5 cm long, absolutely clean, no contamination, incised as well as any surgeon could do with a scalpel. On the lateral side there was a similar horizontal incision a little higher again cleanly incised.

On exploration of the outer wound the bite had gone right through the bone and in doing so, divided 3 extensor tendons. Over the next 2 hours both wounds were debrided for safety's sake, the tendons repaired and the medial wound was then explored. I was amazed to find the medial ligament totally divided. This gives the principle medial support to the ankle and a further extensor tendon had been damaged. In all, over two and a half hours were spent cleaning the wounds and repairing the damage.

It was explained to the patient that as far as we know, that particular wahoo had been eating fairly clean fish of late. With the passage of time and antibiotics there was no subsequent contamination and he went back to Melbourne.