SPUMS MELBOURNE MEETING 20th NOVEMBER 1982

CASES OF SEVERE NEUROLOGICAL DECOMPRESSION SICKNESS WITH VERY DIFFERENT RESULTS AFTER TREATMENT

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This paper contrasts two cases of severe neurological bends and discusses their management. The second, more complicated case, particularly highlights some of the practical problems of management.

CASE REPORTS

The first case was a 26 year old abalone diver. In 1975, he was diving off the South East coast of Australia in depths varying from 80 to 30 feet of water in three separate dives for approximately 170 minutes. He then ascended fairly rapidly from about 75 feet and did not make any decompression stops. Within five minutes of surfacing there was rapid onset of paralysis of both legs and his right arm. Over the next 20 minutes he also experienced abdominal pain, dyspnoea and numbness of the left side of his face.

After lying on the deck of his boat for half an hour, he was helped into his diving gear and lowered to l0 feet. Within a few minutes power began to return to his limbs and his dyspnoea resolved, but he experienced severe limb cramps. He spent 30 minutes at 30 feet, and then ascended, with a 10 minute stop at 10 feet. His boat then brought him inshore, at which stage he was experiencing pain in his left arm and right leg which was becoming progressively worse.

He was immediately placed in a chamber and recompressed to a pressure equivalent to 60 feet of sea water (fsw) and commenced breathing oxygen from the built in breathing system (BIBS) for 20 minutes of every 25 minutes. This recompression was started approximately two hours from the onset of his initial symptoms.

He was subsequently examined by a medical practitioner about one hour after recompression, while still at 60 feet. He was quadriplegic and shocked, with a systolic blood pressure of 90 mm Hg. Two intravenous infusions were started and saline solution given. His bladder was catheterised and found to be empty. He was given Dexamethasone 20 mg intravenously. Once urine production began he was given Rheomacrodex in saline.

He was held at 60 fsw pressure for three hours and continued the oxygen breathing cycles. During this time he regained full movements of all limbs, but remained lethargic. He was subsequently decompressed on a straight profile at 6 minutes per foot without any relapse. He climbed out of the chamber carrying his intravenous infusion bottle. He had a large diuresis over the next six hours.

Careful examination by the medical practitioner involved, two weeks after the incident, revealed no abnormalities apart from moderate hypertension.

Despite medical advice he returned to abalone diving. He was known to have remained free of problems for the next two years, since when his whereabouts are unknown.

The second case is that of a 35 year old marine biologist who was collecting samples off the South East Coast of Australia. In January 1982, he was diving with two colleagues using compressed air.

They descended to between 190 and 200 feet and spent 12 minutes at this depth. The patient made a brief excursion to approximately 210 feet. His companions did not make this excursion. It was not part of the original pre-dive plan. It is likely he was suffering from nitrogen narcosis.

They had planned to ascend according to the US Navy Table for 200 feet for 15 minutes. This requires a 2 minute, 50 second ascent to 30 feet, then a 1 minute stop, with further stops at 20 feet for 4 minutes and 10 feet for 10 minutes.

At 30 feet he felt very fatigued and developed pain in his right elbow. This remained unchanged until after he surfaced. However, within two minutes of surfacing, his elbow pain became very severe and he was unable to lift the arm above his shoulder. Using fresh tanks, he and a companion re-entered the water and did stops of five minutes at 30 feet, 5 minutes at 20 feet and 10 minutes at 10 feet before surfacing. During this his symptoms improved so that on arrival at the surface he was experiencing only a mild ache in his right elbow. However, over the subsequent 30 to 60 minutes he developed paraesthesia in both hands, loss of co-ordination of all limbs and marked lethargy. It was at this stage that medical help was requested.

On reaching land he was given oxygen by face mask and an intravenous infusion started. Later he was flown at wave top level to a barge that was working in Bass Strait. There was a delay of 7 1/2 hours between surfacing and recompression, largely due to problems of communication and transport.

On arrival at the decompression chamber, he was assessed by a medical practitioner. He was almost quadriplegic with only minimal hip movement, absent power to the intercostal muscles, but good diaphragmatic respiration and anaesthesia below the level of the nipples. His cranial nerves were normal. He had peripheral vaso-constriction with a systolic blood pressure of 110 mm Hg.

He was compressed to the equivalent of 60 fsw using the same oxygen cycles as the first case. There was a medical practitioner in the chamber throughout his treatment. Rheomacrodex and Dexamethasone 12 mg were infused. Calcium heparin was administered subcutaneously and Diazepam was given as necessary to control severe muscle spasm. After two hours at 60 fsw, there had been no

improvement. The decision was made to compress further to the equivalent of 165 fsw on air. In retrospect, I think that was a mistake.

The patient was held at 165 fsw for two hours with some improvement in power, but not in co-ordination. We decided to attempt a cautious decompression using US Navy Table 4. However, on reaching 80 fsw, the patient began to deteriorate rapidly.

At this stage, we decided to recompress to the depth of maximum relief of symptoms, using an oxy-helium atmosphere, and maintaining a partial pressure of oxygen of 0.6 Bar. Over the following three hours the pressure was increased in increments of 30 fsw to a depth of 300 feet. There was no apparent improvement in the patient after 270 fsw pressure.

Assessment of the patient shortly after arriving at 300 fsw pressure revealed reasonable power above the waist, but very poor co-ordination. There was almost complete paralysis of the lower limbs, no bladder sensation and complete anaesthesia below the waist. The patient, doctor, and attendant were kept at this pressure for 102 hours. This was an arbitrary time in the hope of avoiding persistence of a gas phase in damaged nervous tissue.

We feel that it is not possible to determine from the published experimental data a time after which one can safely assume that a gas phase is no longer present. Clearly, the longer the time under pressure, the less the risk becomes.

However, there are factors, such as the psychological and medical care of a paraplegic patient in a recompression chamber, that must be considered. We decided to decompress on the profile shown in table one, while keeping a careful watch on the patient for signs of deterioration. Some of the holding periods were timed to allow sleep periods for the patient and physician in order to minimise the risks of missing an early relapse.

The decompression was completed uneventfully. The general condition of the patient was good, compared to his original problem. He had good power above the waist, but still had weakness in the legs, particularly the right and still required bladder catheterisation.

Full assessment suggested a cervical cord lesion at C5 level largely in the right lateral aspect of the cord involving the ascending lateral spino-thalamic tract and the descending right cortico-spinal tract with posterior column involvement bilaterally, as shown in figure 1. This distribution of damage is highly suggestive of the pattern seen in venous infarction with sparing of the grey matter.

His progress since then has been that of a steady improvement. He has returned to work and is able to walk. The main problems remaining are spasticity of his lower limbs and a contracted, small volume bladder.

CONCLUSIONS

There are several points to highlight from these cases. Both demonstrate the practical problems of adequate treatment in divers who have not considered the possibility of needing recompression treatment. I am sure their attempts at recompressing themselves in water made their lesions worse.

The first case demonstrates (as has been recorded many times before) that early recompression on a long oxygen table can give excellent results.

In the second case, there was a 7 1/2 hour delay before recompression. He showed no improvement at 60 fsw equivalent pressure on oxygen for two hours. The decision to compress to 165 fsw equivalent on air may have compounded the problem with further nitrogen accumulation in the tissues. His condition was so serious that to have completed his decompression from that depth could have led to a fatal outcome.

For practical reasons, it was more appropriate for us to convert to an oxyhelium atmosphere rather than use nitrogen-oxygen saturation therapy. We decided that the risks of oxygen toxicity could be kept to a minimum by maintaining the partial pressure of oxygen at 0.6 Bar. When one is faced with a critically ill patient such as this man, one is justified in recompressing on oxy-helium to the depth of maximum relief, if the chamber being used has that capability.

TABLE ONE

300 - 100 feet at the rate of 8 ft/hour
100 - 60 feet at the rate of 4 ft/hour
60 - 00 feet at the rate of 2 ft/hour

Stops were made at:

55 feet for six hours
15 feet for two hours
10 feet for three hours
5 feet for three hours

Total time of treatment - 189 hours, 29 minutes.

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