5. DIPLOMA IN DIVING AND HYPERBARIC MEDICINE

The RAN has formally agreed to support this Diploma by allowing use of SUM staff and facilities for training purposes of prospective candidates. Further discussions are continuing in order to expedite an early start for the course, and more information will be available in the next Newsletter.

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6. CLINICAL CASE REPORT

HISTORY (37 year old Nauruan Islander)

Friday 2nd February

9.30 am Dive to 200 feet for unknown time with stops at 20 feet and 10 feet during ascent (uncertain duration - not following a recognised decompression table).

2.00 pm Dive to approximately 250 feet. Equipment is standard 72 cu ft tank. During this dive air supply was exhausted so diver had to rely on reserve (J valve). During ascent, reserve gas was exhausted and so an immediate return to surface was made.

2.45 pm Arrived at surface. Times may be incorrect, but it is known that tanks are frequently over-pressurised at Nauru so this may explain the length of the dive on a single tank. On leaving the water carrying equipment ashore the diver noticed slight back pain which increased during the next 20 minutes, with some slight associated difficulty in breathing. Over the next few hours, diver noticed some weakness in the legs, severe pains radiating around lower abdomen and to some extent down the legs.

5.00 pm Presented at Nauru Hospital and was admitted.

7.00 pm By this time the patient had developed a total areflexic paralysis of both legs with complete anaesthesia below the xiphisternum. He was given Solu-Cortef 200mg IMI and intermittent O_2 therapy by mask. No other injuries were noted.

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Saturday 3rd February

10.30 am Call for assistance received from Nauru.

1.00 pm Medical officer from SUM discussed problem with doctor on Nauru (poor communications). A diagnosis of spinal decompression sickness was made and the following treatment was advised:

1. 100% O_2 by mask
2. increase steroids
3. Set up IV infusion
4. Indwelling urinary catheter

Arrangements were made with the RAAF to fly to Nauru to bring the diver to HMAS Penguin.

6.00 pm Hercules departed from Richmond.

12.00 midnight Refuelling stop at Port Moresby.

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Sunday 4th February

7.00 am (Sydney time). Arrived at Nauru. Examination of the patient revealed that he now had brisk reflexes in both legs, flexor plantar responses but the paralysis and anaesthesia were still present. His abdomen was soft, slightly tender centrally, however movement particularly sitting up caused intense pain. Upper recti muscles contracted voluntarily. Abdominal and cremasteric reflexes absent.

The patient was placed on the aircraft and flown back to Sydney. Treatment in flight consisted of intravenous fluids, Dexamethasone 4 mg 6th hourly and 100% O_2 by demand valve.

6.00 pm Plane arrived in Sydney and patient transferred to SUM, HMAS Penguin, where he was reassessed. Neurological examination was essentially unchanged except for clonus in both ankles, the left greater than the right. Examination of the chest revealed diminished air entry in all areas with widespread inspiratory and expiratory rhonchi. Vital capacity was 2.35 litres although this appeared to be limited by his abdominal pain. There was also an occasional irritative cough. Respiratory rate was 16/minute.

Prior to entry to the chamber he was given Lazix 40 mg IVI, Aminophylline 250 mg IVI stat and Aminophylline 250 mg and Dexamethasone 4 mg were added to his infusion of 4% Dextrose and N/5 Saline (IL 8th hourly).

8.00 pm Patient placed in RCC approximately 51 hours after his last dive.

Initially compressed to 30 feet (breathing 100% O_2) and after 2 minutes the sensory level dropped to just above the pubis.

After 10 minutes there was a return of sensation to both legs. This included pinprick (dulled) and light tough, position and vibration sense but equivocal 2-point discrimination. There was no return of motor power.

The chamber was then pressurised to 60 foot (20 min O_2/5 min air). After 45 minutes there was no further change and he was then compressed to 165 feet 40% O_2. Again no improvement. He was then decompressed at 6 ft/min to 60 ft and subsequently brought out on the long O_2 table (Table 6B).

Except for the initial improvement there was no further change and in particular his VC remained unaltered.

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Monday 5th February

4.00 am Patient left recompression chamber. At this time a flicker in his thigh muscles was noted and a very small flexion of the knee joints with contraction of hamstrings. There was no ankle or toe movement. Oxygen therapy was stopped because of his pulmonary signs, and further possible hyperbaric therapy was delayed. IV Aminophylline and Dexamethasone were continued and he was also given 500cc of Rheomacrodex over 4 hours. Oral Ampicillin was commenced to combat possible urinary tract infection. Elementary physiotherapy in the form of passive joint movements was commenced.

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Tuesday 6th February Condition unchanged except rhonchi now in anterior upper zones only and his VC was 3.1L. No voluntary movements occurred in lower limbs although there was a marked withdrawal response to a sharp stimulus to the soles. Serum electrolytes and urea were normal. Enzymes SGOT 58 (normal >28 IU), LDH 330 (normal > 50-170 units), and CPK 270 (normal 0-50 units). Chest x-ray and abdomen x-ray NAD. However x-rays of lumbar spine showed old probably fracture of upper anterior lip of L4. There was no history of injury.

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Wednesday 7th February On awakening patient was noticed flexing left leg and to a lesser extent right ankle. This could not be reproduced voluntarily, however a small amount of tone was noted in both thighs at times.

Transferred to RNSH Spinal Unit.

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Wednesday 14th February Bladder remained atonic. Paralysis persisted both legs.

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Wednesday 21st February Some voluntary movement both thighs noted. Also some plantar flexion of ankles (L > R). Bladder atonic.

March 10 Able to walk with assistance of parallel bars. Regaining some functional control of bladder.

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March 15 Able to walk with walking sticks, steady gait, slight (R) sided foot drop.

Bladder residual volume 80ml with functional control.

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March 18 Patient returned to Nauru.

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Some weeks later another patient arrived from Nauru (whose story will be related in the next issue of this Newsletter). This diver reported that our previous patient was no walking well and was seen occasionally to drop his walking sticks and walk down the beach unaided.

COMMENT

After such a long period of time had elapsed, it was doubtful that recompression would be of great benefit to this man. However, the sensory return in the first 10 minutes was quite dramatic.

Whether this improvement was due to hyperbaric oxygenation of ischaemic and oedematous tissue or to shrinkage of a remaining bubble is open to conjecture.

After this time it is likely that no bubble as such was present and this is partially supported by the lack of response at 165 ft.

The only other improvement initially was noted immediately after being removed from the chamber.

Rheomacrodex and steroids did not appear to alter the course over the next 2 days.

Further sessions of hyperbaric O_2 may have a place in management to reduce oedema of the spinal cord, and may ‘swing the balance’ for cells whose O_2 supply is precarious. However, in this case, further HPO sessions were decided against because of the probable pulmonary O_2 toxicity.

The spinal lesion was probably around the T7 level.

Although immediate recompression is the treatment of choice in DS it is felt that even after a long delay recompression is still worthwhile. It is impossible to say at what point in time the benefits are due to recompression and therapeutic decompression (ie. getting rid of a bubble) or due to hyperbaric oxygenation of ischaemic tissue. In the individual case the argument is largely academic, especially

if one routinely uses O_2 tables in treatment.

This case once again illustrates the consequences of diving gear being made freely available without associated training in its use and the limitations and hazards of diving. This man had heard of ‘bends’ and knew one had to stop on ascent before reaching the surface but had never used or even seen any recognised decompression tables. The reason given for being at 250 ft was ‘chasing a little red fish’. A second case of spinal decompression sickness from Nauru several weeks later showed that it is difficult to get the message across.

7. FINANCIAL MEMBERS * * * * * * * * * *

The following are financial members to 30 June 1973:

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Mr F Ashmore
Australian Museum (Dr Frank Talbot)
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Dr V Brand
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