MANAGEMENT OF DELAYED CASES OF DECOMPRESSION SICKNESS—3 CASE REPORTS

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SYNOPSIS

Decompression sickness may give rise to a variety of symptoms and signs. Paralysis or paresis is the common presenting complaint because the victims, usually fishermen divers, ignore "minor" signs and symptoms. Three cases of decompression sickness were treated by therapeutic recompression using hyperbaric oxygen in September/October 1972 at the SAF Diving Centre. Only one session of therapeutic recompression lasting 285 minutes was carried out on each patient because of satisfactory results. The patho-physiology and symptomatology of decompression sickness and the rationale of using hyperbaric oxygenation in therapy are discussed.

INTRODUCTION

Decompression sickness includes any abnormality which is a direct result of too rapid reduction in tension of gases dissolved in the body. Aviators, divers and Caisson workers are commonly exposed to this danger. Medical and paramedical personnel working in hyperbaric conditions may be similarly affected.

In Singapore most of the cases seen are amateur fishermen divers. These people have had no formal training in diving and are ignorant of diving procedures. In the past these cases were treated by the Royal Navy at Naval Base, Sembawang. Today the Ministry of Defence has taken over this responsibility. One common feature of these cases is that they tend to present very late for treatment because their fishing grounds are in the Indian Ocean and the South China Sea.

CASE REPORTS

3 delayed cases of decompression sickness were treated at the SAF Diving Centre, Sembawang, within the months of September/October 1972.

Case 1

C. T. H. a 45 year old male Chinese. The patient went diving on 23 Sept. 72 in the South China Sea. He claimed to have dived 3 times to 60 feet for 30 minutes each time using compressed air. He did not undergo any decompression stop. On surfacing after the last dive he felt numb in both lower limbs and became very weak. A short while later he experienced a severe pain in his left shoulder. It took his boat 3 days to return to Singapore. During this period he could not open his bowels or micturate. He was catheterised three times by his colleagues. He was not able to walk. There was no loss of consciousness.

Physical examination done on 26 Sept. 72 before the start of treatment revealed that the power of the right lower limb was grade 1 and the left grade 2. There was bilateral loss of pain sensation below the level of T10. Knee and ankle jerks were brisk and plantar reflex was upgoing. There was complete loss of bladder and bowel functions.

The patient was recompressed to 60 ft. and treated on a therapeutic decompression schedule of oxygen and air for a total time of 285 minutes.

Examination after recompression treatment revealed that both his lower limbs had improved considerably in power. His right lower limb improved to grade 3 and left, grade 4. The loss of pain sensation shifted to the L3 level on the left side but there was no change in the reflexes or the bladder and bowel control. Patient was again examined three weeks after recompression therapy. His left lower limb had recovered fully in power while his right lower limb was grade 4 in power. He was able to walk normally. The loss of pain sensation had fully recovered on his left side while on the right it had shifted to L3 level. His bowel and bladder functions were fully recovered.

Case 2

Q. T. M. a 21 year old male Chinese. He dived to 70 feet—3 times on 16 Oct. 72 in the South China Sea for a total duration of 90 minutes. No decompression stops were done. Half an hour after his last dive he felt very tired with numbness in both feet. Two hours later he found that he could not move his lower limbs or pass urine. He was then put into the sea with his diving gear to 30 feet for 2 hours. On surfacing he could move his lower

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limbs a little but was not able to stand up. He was catheterised by his colleagues. He returned to Singapore on 22 Oct. 72 and was referred to us on 23 Oct. 72.

On examination before treatment, findings were confined to the central nervous system. The right lower limb was grade 3 in power and the left lower limb grade 4. Patient was unable to stand up on his own. Hypoaesthesia was evident below L2 bilaterally. Knee and ankle jerks were brisk, plantar reflex was downgoing. Bladder and bowel control were normal.

Patient was recompressed to 60 ft. followed by slow decompression on oxygen/air for a total of 285 minutes.

Examination after the treatment showed that the right lower limb had improved to grade 4 in power and the left lower limb grade 5. Patient was able to stand up and walk unsteadily. There was no change in the level of hypoaesthesia.

Case 3

K. N. S. a 37 year old male Chinese. He made a number of dives on 16 Oct. the details of which he could not recall. The next day he dived to 120 feet twice for half an hour each time. There were no decompression stops for any of the dives. Ten minutes after the last dive he felt giddy and experienced pain in both shoulders. He was returned to the water to 30 ft. He soon became unconscious and was found floating on the surface.

After recovery he was again returned to the water where he stayed at 30 feet for 4 hours. His lower limbs became numb after one hour. When he was finally returned to the surface both his lower limbs were paralysed. There was no more pain in his shoulders. He could not pass urine and was catheterised three times by his colleagues during the trip home. He recovered some power in his lower limbs but numbness persisted. He arrived on 18 Oct. and underwent recompression therapy on the same day.

Examination before treatment revealed the power of the right limb to be grade 2 and the left, grade 3. The patient was unable to stand up on his own. There was bilateral loss of pain sensation below T12. Knee and ankle jerks were brisk and the plantar reflex was upgoing. He had no control of his bladder and was dribbling urine.

Patient was recompressed to 60 feet and slowly decompressed on oxygen/air for a total of 285 minutes.

Examination after treatment revealed an improvement of power of the right lower limb to grade 3 and the left, grade 4. The patient was able to stand up on his own but not able to walk. There was no improvement in the anaesthesia below T12. There was no change in the tendon jerks and plantar reflex. The patient recovered control of his bladder and passed urine while in the chamber.

Examination two months after treatment revealed further improvement. The patient was able to stand up from a squatting position and walk. The power of the right lower limb improved to grade 4 and the left, grade 5. His sensory function improved and sensations were normal on the left side with hypoaesthesia below T12 on the right. He had control of the bladder in the day but suffered from incontinence in the night.

DISCUSSION

Bubble Formation

When the rate of decompression is too rapid the quantity of gas in the blood being liberated will exceed the physiological mechanisms for coping with it and bubbles will form. One peculiar feature is that if a group of men were decompressed together after the same exposure to pressure most would be unaffected while a few might become severely affected. Such differences in individual susceptibility could be due to the random nature of bubble formation or a variation of individual tolerances to the bubbles which may be formed after every decompression.

Bubbles may form in intra and extra vascular tissues. The extravascular tissues involved may be extracellular (synovial fluid, C.S.F.) or intracellular (fat tissue, liver and spinal cord). Bubbles in fat cells may result in rupture of the cells with release of active agents causing local haemorrhage and release of gas and fat emboli into the circulation. Intravascular bubbles are more commonly found in the veins because of its lower blood pressure.

It is difficult to decide on the site of origin of intravascular bubbles. Bubbles are more frequent in veins draining fatty tissues and muscles of active limbs. In animals decompressed from great pressures (>10 ATA) bubbles appeared in both veins and arteries (Gramenitskii and Savich, 1965).

Post mortem examination of severe cases of decompression sickness have revealed intravascular bubbles, inflammation and softening in the brain and spinal cord. The possible mechanism is the reduction of blood flow by embolic bubbles leading to hypoxia, oedema and infarction and the loss of neuron function due to distortion by an expanding bubble.

Factors which predispose to decompression sickness include exercise, obesity, dehydration, cold and injury. On the other hand a greater tolerance

to the effects of decompression is built up by an individual who has dived regularly for a number of years.

Symptomatology

The symptoms presented by the 3 cases reviewed were numbness and paresis of lower limbs in all 3 cases. One case reported pain in left shoulder while another case reported pain in both shoulders. Bowel and urinary systems were affected in all 3 cases.

For the most likely reason of ignorance, it is to be expected that cases of decompression sickness with milder symptoms do not present for treatment. On the other hand, we have heard of cases from fishermen divers that some have died after refusing to present at the hospital for treatment even with serious complaints.

Rivera (1964) in his analysis of 935 cases of divers treated at the US Navy reported the following frequency of symptoms:—

Localised pain 91·8%
Numbness, paraesthesia 21%
Muscular weakness 21%
Rash 15%
Dizziness 8·5%
Visual disturbance 6·8%
Paralysis 6·1%
Headache 3·9%
Loss of consciousness 2·7%
Urinary dysfunction 2·5%
Dyspnoea (chokes) 2·0%
Fatigue 1·2%
Convulsion 1·1%
Oedema 0·5%

It is noted that in all the 3 cases, the symptoms presented within the first hour of the last fateful dive. Rivera in the same series reported the following onset of symptoms in relation to time.

During decompression 9·5%
within 1 hour 57·2%
1 - 2 hours 69·8%
3 - 6 hours 90·2%
7 - 12 hours 97·2%
13 - 24 hours 99·7%
24 - 36 hours (1 case)

A very significant finding in the 3 cases is the attempt at treatment by water recompression therapy. In 2 out of the 3 cases it had apparently improved the clinical picture while one was made worse. The residual picture remained clinically the same during the long journey home till they were treated by Hyperbaric Oxygen Therapy.

Worthy of note is the possession of urethral catheters by these fishermen divers. Experience has taught them that this is a useful item in the event of 'bends' with paralysis of the bladder. Investigations into urinary infection and urethral and bladder trauma are therefore necessary in such cases.

The progress of all 3 cases can be said to be spectacular taking into consideration the delay in treatment. The remaining weakness, patches of numbness, and difficulty with bowel and bladder control will be likely to remain permanently if they persist for a period longer than 3 - 4 months.

Treatment and Management

The "oxygen table" was used in all three cases. The patients breathed 100% oxygen. They were maintained at 60 feet (2·8 ATA) for a total of one hour after which they were decompressed slowly taking a total of 285 minutes. Air was breathed intermittently to prevent oxygen toxicity.

After the therapeutic recompression treatment, the patients were placed on 100% oxygen breathing for 24 - 48 hours with regular intervals on air breathing to prevent chronic oxygen toxicity effects on the lungs. They were given physiotherapy for a few months. A careful watch for urinary infection was necessary because patients were catheterised by amateurs before presenting for treatment.

Rationale of Using Pure Oxygen at 60 Feet

Symptoms that are refractory to treatment are usually due to delay in presenting for treatment. Tissue rendered hypoxic by ischaemic action of bubble emboli and extracellular oedema cannot usually be resolved solely by reapplication of high pressure.

When a person breathes air at 1 ATA the haemoglobin is approximately 96% saturated with oxygen but there is 2·36 ml. of oxygen in true physiological solution in 100 ml. of blood. Breathing pure oxygen at 3 ATA allows complete saturation of haemoglobin as well as some 7·08 ml. of oxygen to dissolve in 100 ml. of blood. This dissolved oxygen is readily available to the hypoxic tissues (being supplied by the process of diffusion or by collateral channels). An oxygen filled lung will also greatly increase the gradient of nitrogen flow from the tissues to the capillaries and hence to the alveolar spaces resulting in a faster desaturation of nitrogen in the body. High pressure oxygen therapy then, is a more specific therapy than the conventional method of recompression to 165 feet (6 ATA) on air which depends more on the physical action of pressure on the bubbles.

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