Discussion of a case

Dr Dries Jones
(Senior Medical Officer, SAN Medical Centre, Simonstown, RSA)

(Reprinted by kind permission of BAROLOGIA).

Diver GJE, age 19, performed a controlled ascent during his training in September 1975. This followed 18 minutes at 21.3 metres (70 feet). Within minutes of surfacing he developed an acute headache, pain in the right side of his chest, partial blindness, and became unconscious. He was immediately put onto 100% oxygen and transported to the SAN Diving School. On arrival, within 10 minutes, he was noted to be disorientated and to have a severe headache.

Therapeutic recompression was initiated according to Table 62 (Oxygen at 18 metres. Total time 4 hours 45 minutes). Disorientation, chest pain and blindness had disappeared after 5 minutes at 18 metres. Pulse 60/min, BP 120/80, ENT:NAD. Fundoscopy normal. Respirator system: NAD. CNS-absent abdominal reflexes, hyper activity in patella reflexes and hypaesthesia of the lower limbs. Severe headache persisted and vomiting started after 30 minutes.

The headache and vomiting persisted even after 3.5 hours. A blood-sugar evaluation revealed blood glucose of less than 40 mgm% so 20cc of a 50% Dextrose solution was given intravenously, with immediate relief of both headache and vomiting. After 4 hours 45 minutes he was symptom free. Mild weakness was felt in the legs but could not be demonstrated. A full clinical examination revealed no abnormality. X-ray of the chest was normal. Diagnosis: pulmonary barotrauma, with arterial air embolism either aggravated or simulated by hypoglycaemia.

Four hours later the patient suddenly collapsed with acute headache, becoming unconscious with flaccid paralysis of the lower extremities. He was placed onto oxygen and taken down to 18 metres. Roused with difficulty, but then disorientated. Still flaccid paralysis of lower legs with complete sensory fall-out below the waist and absent abdominal reflexes. Level of consciousness improved and he could move his legs with difficulty after 1 hour. On this occasion intravenous dextrose did not improve the patient's condition.

On advice from the UK the patient was taken down to a depth of 50 metres for treatment according to Table 55, after 2 and a half hours at 18 metres breathing oxygen intermittently. On arrival at 55 metres again, complete paralysis of legs with pains in legs and worsening headache. Medical treatment was started after half an hour at 50 metres:

Dextran 40 ivi — Heparin 5,000 U subcutaneously
Dexamethazome 100 mgm 8 hourly
Furasimide 20 mgm ivi
Maxolon 2 cc statim.
After 2 hours the patient could stand with difficulty.
After 3 and a half hours symptoms of urine retention, but due to exposure by all doctors, a catheter could only be introduced 2 hours later (at 18 metres).

After 2 hours at 18 metres the patient developed an acute pain in the right side of his chest, with paralysis of both legs. The breathing mixture was then changed to 100% for 20 minutes and air for 5 minutes for the third hour and again for the sixth and last hour of this stop. The symptoms cleared whilst on the first 20 minutes of oxygen. The same regime was followed the hour before and after depth changes from 18-15, 15-12 and 12-9 metres. From 9-6 metres the depth change

was made on oxygen for 1 hour. At the 6 and 3 metre stops, air and oxygen for 1 hour stops alternately without shortening decompression (as in Table 54) has been used due to the seriousness of the symptoms.

Seeing that the last symptoms and/or signs were found 24 hours before surfacing, it came as quite a shock when the patient had difficulty in walking due to weakness of hip muscles, and to some extent also weakness in the left leg. He also had some sensory fallout in the right leg in the segmental distribution of the L4-S5. However a full neurological examination after 3 months revealed no neurological residuae and the diver was declared fit to dive again.

Discussion

1. Diagnosis
2. Therapeutic decompression: air or oxygen
3. Medical Treatment
4. Advice re diving fitness
5. Prevention: a) training
   b) Diagnosis of tendency

1. Diagnosis

The maximum no-decompression dive time at 70 feet (21.3 metres) on US air tables is 50 minutes, and on RN air tables 40 minutes. The dive time in this case was 18 minutes. In questioning the diver after the incident it seemed that he started following a small air bubble by holding his thumb underneath it. As the bubble expanded on its ascent he admitted that he had difficulty in keeping up with it. It can only be surmised that he had closed his glottis for the crucial time to achieve a pressure of 90/150mm Hg=1, 18-1,97 msw to cause the over-distension to produce lung damage. There is also the possibility that a mucus plug, etc could have caused obstruction of one of the smaller bronchi, this causing the injury, ie. rupture of alveoli with air bubbles being sucked into the pulmonary veins and so being distributed to the systemic circulation, causing arterial air embolism. The diagnosis this seems to be pulmonary barotrauma with arterial air embolism causing cerebral and spinal cord emboli.

According to Spencer (1976) any excess inert gas stressed the nucleation sites where bubbles are formed. One can thus assume that the introduction of air from the pulmonary bed may have caused these nucleation sites to grow and rupture causing secondary bubbles, depending on how critically they were stressed before the time.

The concept of bubble/blood interface effects causing protein denaturation, formation of lipid emboli, red-blood cell sludging in micro-vessels, platelet aggregation and activation of both coagulation and fibrinic systems has become widely accepted. So the initial release of small quantities of gas from a few over-distended alveoli may be the trigger for severe and life-threatening symptoms developing.

2. Therapeutic Decompression Air or Oxygen

The treatment of pulmonary barotrauma with air embolism has empirically been on compressed air at 50 metres. The argument is: at 50 metres (6 ATA) there will be a reduction in bubble diameter as volume is reduced to one sixth, and this will allow the bubble to pass through the capillary to the lungs, or to a point lower down, where it would cause few symptoms. This argument is based on spherical bubbles. According to Buckles (1968) the intravenous bubbles have

a length-to-diameter ratio varying from 1-1 to 1-30. Bubble diameter reduction seems to be beneficial only under certain circumstances. For this possibly beneficial effect it was proposed by Walder (1967) and the Bureau of Medicine and Surgery Instruction (1976) to take divers with low inert gas levels down to 50 metres for a short time and then bring them back to 18 metres to complete one of the therapeutic oxygen schedules.

The beneficial effects of oxygen at 3 ATA as compared to air at 6 ATA are:

a. To keep the partial pressure of nitrogen as low as possible, to minimise any contribution which absorption of nitrogen during the recompression itself may make to the recurrence of the lesion.

b. Studies by Wyman and Van Liew indicated that the lifetime of a bubble should not vary appreciably with pressures in excess of 3 ATA.

c. The bubble reduction of an oxygen breathing patient is 4-5 times greater than with air breathing at 3 ATA.

d. The gas tension gradient from bubble to tissue is maintained optimally throughout recompression, therefore unlikely to permit bubble growth with re-occurrence of symptoms.

e. Oxygen breathing at increased pressure is specific treatment for adequate oxygenation of hypoxic tissue, preventing further oedema with an increased hypoxic area.

f. It has further been shown that oxygen at 3 ATA is a specific treatment for cerebral oedema (Thiede and Mahley 1976).

One incident in which the use of oxygen tables seems to be contra-indicated is after long deep air dives where the tissue pp of N_2 would be such that a pressure of 3 ATA would not arrest bubble growth.

The recurrence of symptoms in this case could have been attributed to:

1. The presence of blood/gas interface action with haemoconcentration, red cell aggregation, increased clotting tendencies with development of disseminated intravascular coagulation.

2. Inadequate time under pressure

    a. Workman himself advocated the lengthening of the Table 62 by adding an extra 5 minutes air and 20 minutes on oxygen at 18 metres, an extra 15 minutes air, 60 minutes oxygen at the 9 metre stop, or to include both, in slow responding patients.
    b. Perhaps if Table 54 or 55 had been followed, there might have been no lapse.

3. Medical Treatment

This should always be supportive to therapeutic decompression and should be used alone only when no recompression facilities are available. There is, however, more and more evidence that in all cases of acute decompression sickness concurrent medical treatment is mandatory (Elliot 1974 COMEX Medical Handbook; Boorman 1968 et al). Medication as mentioned has been advocated for many years not only for decompression accidents but also as treatment of cerebral oedema. Heparin seems to be more effective in smaller doses, ie. 2000 U given as a bolus intravenously, 6 hourly. The dose of dexamethazone seems to have been

unnecessarily high: after an initial dose of 100 mg, 8 mg 8 hourly seems to be sufficient. Steroids used in conjunction with hyperbaric oxygen have been reported to increase the likelihood of CNS toxicity. The use of Diazepam prior to the start of decompression should overcome this problem. Glucose 50%, apart from the value of its use in hypoglycaemia, has a direct effect in the reduction of intracranial pressure. Unfortunately a rebound phenomenon occurs due to its rapid metabolism, this causing hypo-osmolarity with subsequent increase in brain water. When used for hypoglycaemia it should be given in saline. The question arises whether our use of 50% Dextrose had well given the initial relief of symptoms by its effect on the brain oedema, but caused a relapse through causing brain oedema afterwards.

4. Advice regarding diving fitness after a decompression incident.

   a. It has been shown by Elliot and other researchers that changes in the blood composition take up to 10 days before returning to normal levels. It thus seems wise to prohibit diving during this period.

   b. If any neurological residue persists after 6 months the diver should be advised to stop diving altogether, due to an increased susceptibility to future damage by an already hampered nervous system.

5. Prevention of accidents.

   a. Training in all emergency procedures in diving should be performed so regularly and so strictly that it becomes a second nature.

   b. Diagnosis of emergency procedures should be the first priority of the examining MO. The Doppler system using ultrasound to pick up bubbles after supposedly safe dives could in the future provide help in discovering, for exclusion, bends-prone divers.

References

1. Spencer MP. Decompression limits for compressed air determined by Ultrasonically detected bubbles. *Journal of Applied Physiology*. 1975; 40(2): 229-235.

2. Buckles GR. The physics of bubble formation and growth. *Aerospace Medicine*. 1968; 39(39) Oct, 1062-1069.

3. Walder DN. Decompression of Compressed Air Workers. McCallum, *Civil Engineering*. UK, Oriel Press; 1967: 13

4. Bureau of Medicine and Surgery *Instruction* 6420.2 of 22 August 1967

5. Thiede and Manley. *Aviation, Space and Environmental Medicine*. 1976; May: 553-555.

6. Ayman, Scholander, Edwars and Irving. On the Stability of Gas Bubbles in Sea Water. *Journal of Marine Res*. 1952: 47-62.

7. Van Liew HD. Factors in the Resolution of Tissue Gas Bubbles. Williams and Wilkins. Underwater Physiology. Baltimore, 1967.

8. Elliot DH. Acute Decompression Sickness. *Lancet*. 1974; 16 Nov: 1193-1199.

9. Comex Medical Book II. Comex Diving Ltd, Bucksburn, Aberdeen.

10. Bormann RC. Limitations in the Treatment of Diving and Aviation Bends by Increased Ambient Pressure. *Aerospace Medicine*. Oct 1968: 1070-1076.