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Medical Aspects of a Simulated Dive to 1500 feet (458 metres)

Each attempt in the history of diving to progress to greater depths has been met by a succession of apparently insurmountable barriers. The early problems were overcome by improved engineering and the later ones by a better understanding of the physiological mechanisms involved.

The most recent apparent barrier, now called the high-pressure nervous syndrome (HPNS), was first observed in 1964 at the Royal Naval Physiological Laboratory. Following compression to 600 and 800 ft (183 and 244 m) men developed a coarse tremor and muscular incoordination which ameliorated while at depth.

In 1968 Brauer and Delauze at Marseilles abandoned a simulated dive at 1,190 ft (363 m) after 4 minutes at that depth because of the HPNS, which was described as gross muscular incoordination and drowsiness, with a tendency to sleep if not occupied. The EEG records of the divers showed greatly increased theta activity (6 Ho) and slow waves, which animal experiments had shown might be pre-convulsive. Because of these findings, Brauer postulated the existence of a 'helium barrier'. The existence of this barrier was questioned after a Swiss dive at the Royal Naval Physiological Laboratory (RNPL) to 1,150 ft (351 m), only 40 ft (12 m) shallower than the Marseilles -dive, had produced no similar problems (Buhlmann et al. 1970).

It became important to investigate whether or not the 'helium barrier' was a definite entity and to assess the safety factors for operational diving, expected soon down to 1,000 ft (305 m). Two men were selected from the volunteers from RNPL for the final dive, both qualified divers and experienced in pressure work; A, aged 26, and B, aged 20.

Environmental Details

The chamber with an airlock was 16 ft (4 9 m) long and 5 ft 6 in (1 68 m) in diameter, and additionally there was a small handlock for the passage of food, etc.

Po2 was maintained at 045 bar as a compromise between avoiding the chronic toxic effects caused by Po, in excess of 0-6 bar and the apparent hypoxia at high pressure with Po. of 021 bar described by Chouteau et al. (1967).

The atmosphere was continuously drawn through two gas scrubbers for the removal of carbon dioxide, water vapour and atmospheric contaminants.

Because the high thermal conductivity of helium raises the lower limit of thermal comfort by about 10°C and narrows the range for comfort, the temperature had to be maintained at 29 -310C.

Investigations and medical monitoring

The medical supervisors were in no way involved in the scientific investigations. Before the dives each subject was medically examined and subjected to radiographic examination of chest and long bones.

During the dive the subjects carried out many performance and physiological tests which occupied them for 7 hours each day and included exercise on a reclining bicycle ergo meter. Among the investigations were helium tremor tests, respiratory function tests and blood and urine analysis.

The ECG and EEG recorded by the investigators were available to the medical officers on both oscilloscope and paper print-out. There was continuous recording of the ECG but the EEG and its frequency analysis were only recorded during investigations or when required.

The difficulties of communications with divers in helium are well known, so the speech unscrambler built by Dr J Gill of the Admiralty Research Laboratory was of inestimable value, especially with the problems encountered in the decompression phase.

Compression phase

To permit study of HPNS a compression rate of 100 ft (30 5 m) in 6 minutes was used in this dive but broken into stages to permit adaptation and recovery (Fig 1). Much faster rates are known to produce gross muscular tremor while slower rates produce a lesser effect.

The responses to pressure of the 2 men were dissimilar in character (RNPL Report IR 1/70). On EEG, Diver A, who showed a dominant theta activity at the surface, showed a decrease in all frequencies from 600 ft (183 m) down, and this decrease reached its maximum at 1,000 ft (305 m). However, with each compression phase he developed very marked muscular tremor.

Diver B showed a considerable increase in theta activity which was stimulated by each compression phase, reaching a peak some 6 hours after attaining a new depth and returning to near normal levels over the next 12 hours. The theta activity showed no further increase beyond 1,000 ft (305 m). Diver B showed very little muscular tremor when compared with A, but following compression to 600 and 1,000 ft (183 and 305 m) he complained of nausea and dizziness.

Decompression phase

Dives of these durations, where the inert gas tension of the tissues reaches equilibrium with the inspired gas, are called saturation dives. Once saturation at any given pressure has been reached, any further length of time may be spent at that pressure without apparently increasing the time required for decompression, which at 1,500 ft (458 m) is considerable.

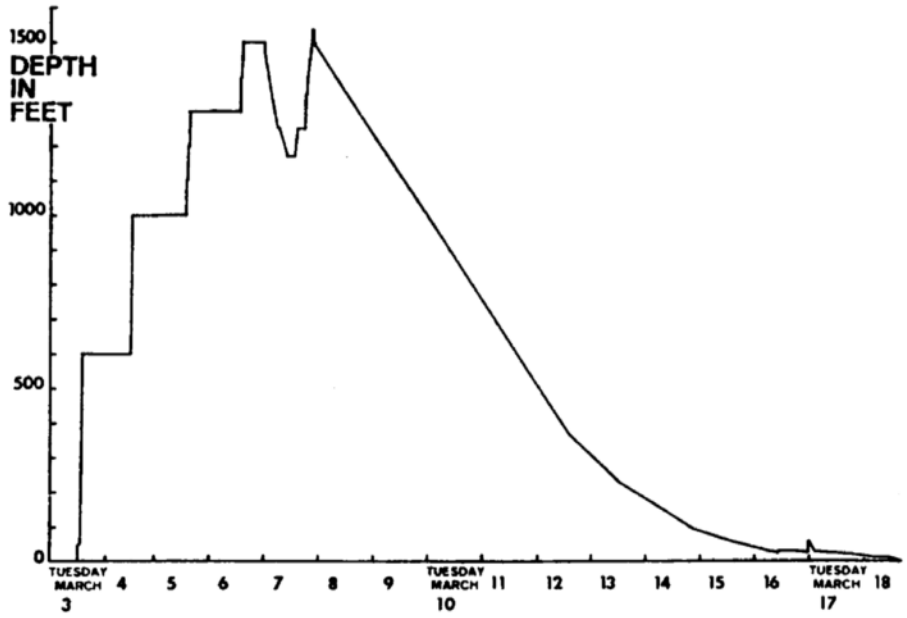

Fig 1 Simulated dive to 1500 feet (458 metres)

From experience with previous short dives as deep as 800 ft (244 m), and the treatment of a number of decompression sickness cases occurring at depth, an approximate decompression schedule for this dive was devised and used in the form shown in Fig 2.

This form of decompression with k=0-052 had been successfully used in the treatment of men with bends occurring as deep as 455 ft (138-8 m) and also for a 24-hour dive at 100 ft (30 5 m) in the preliminary dive series. Subsequent dives showed, however, that the value of k had to be reduced with increasing depth.

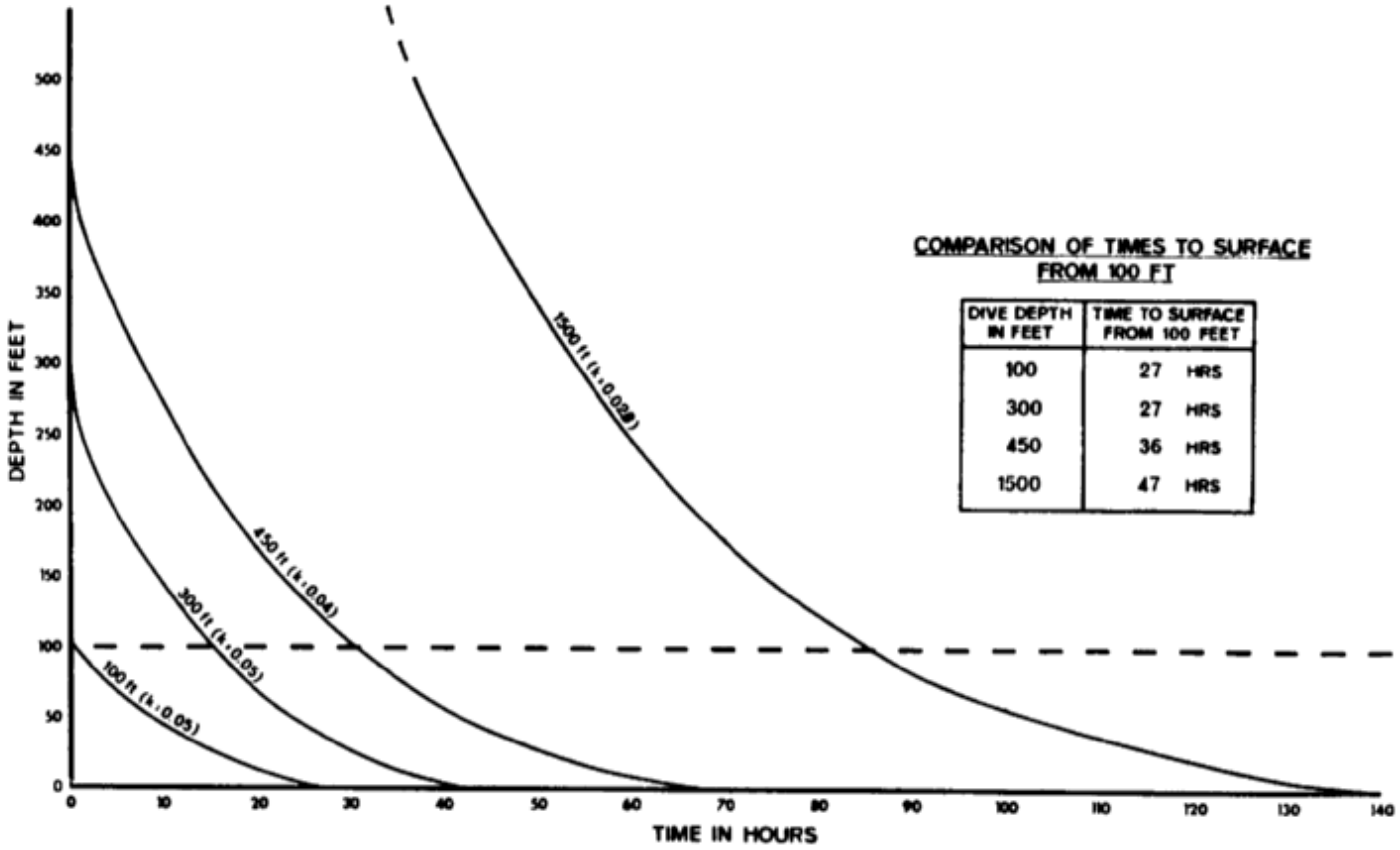

Fig 2 Comparison of decompression in the 1500 ft (458 m) series from 500 ft (152 5 m) to surface. Formula of decompression: Y = Y.e -kt where Y =pressure at time t, YO = pressure at start of decompression, t = time in hours, k = constant

Therefore, k was reduced to 0-028 for the exponential bleed used in the decompression from 1,500 ft (458 m), which began at 23.00 hours on 6.3.70 with an initial reduction in pressure of 1 bar (33 ft; 10 m) (Fig 1). At 1,280 ft (390 m), Diver B's ECG began to show irregular low voltage complexes and occasional negative waves, while his EEG showed increased delta activity. These findings coincided with the onset of nausea and dizziness when he opened his eyes, about which he only complained an hour later at 1,260 ft (384 m). He was observed to have nystagmus and a tendency to fall to the right and he vomited several times. He improved after an hour, so decompression was continued, but three hours later, at 1,175 ft (358 4 m), the symptoms

recurred, accompanied by some deafness in the right ear. The decompression was stopped and Diver A, guided from outside, demonstrated a negative Weber test and a 30% increase in tremor on Diver B's right side; at this time the lesion was diagnosed as a labyrinthine bend. The subjects were slowly recompressed to 1,250 ft (381-3 m), but two hours later Diver B had an exacerbation of symptoms so recompression was continued to 1,350 ft (411-8 m).

Recompression was continued in 50 ft (15 25 m) stages until a maximum depth of 1,535 ft (468 m) was reached. At this depth Diver A developed HPNS and his EEG showed considerable slow wave activity. The pressure was immediately dropped to 1,500 feet (458 m) and decompression begun at 10 ft (3 05 m) per hour.

The condition of both divers improved while decompression continued with some early transient niggles until 16.3.70 (26 ft; 7.9 m). Diver A was seen to be rubbing his right thigh and on questioning confessed that it had been aching for three days, but was now worse and interfering with his sleep. A 4 ft (1-2 m) pressure drop exacerbated the pain so he was recompressed. Initially his symptoms worsened and an oxygen therapy was unsuccessfully superimposed at 60 ft (18-3 m). Eventually the decompression was continued when his symptoms had been ameliorated by analgesics and they surfaced at 16.05 hours on 18.3.70. Post-dive medical examinations showed no abnormalities beyond slight nystagmus to the right in Diver B and a weight loss of less than 8 lb (0-363 kg).

Discussion and Conclusions

Medical: It is now 10 months since the dive and there is no radiological evidence of aseptic bone necrosis in either man.
Dr R R A Coles of the Institute of Sound and Vibration, Southampton, has examined Diver B twice since the dive. Hearing was normal, but there was some spontaneous nystagmus indicating impairment of the right side and a severe right canal paresis, presumed to be due to a 'labyrinthine bend'. There has been no change from the post-dive examination except that he has adapted well to his vestibular imbalance. It is possible that this lesion could in some way be related to an episode of severe acoustic trauma which occurred to his right ear at the start of the dive.

Compression

During the initial compression, an increase of 200 ft (61 m) from 1,300 ft (397 m) to 1,500 ft (458 m) in I h 12 min (including a one-hour halt half way) was uneventful. However, during the recompression therapy, moving 285 ft (87 m) from 1,250 ft (381 m) to 1,535 ft (468 m) in 4 h (with no significant pause) caused Diver A to develop HPNS. Two differences are apparent immediately betwoen the two compressions which could be responsible for the different results: the first had a 1-hour halt half way and the second was 35 ft (10-7 m) deeper. Other intangible factors such as stress could have played a part.

In the light of more recent knowledge it seems unlikely that the greater depth was the cause of Diver A's problems. The HPNS is undoubtedly rate-sensitive but it appears that unless time at intervals is allowed for recovery the effect of compression accumulates to a potentially dangerous degree. It seems that with the right type of compression there is no helium barrier at 1,200 ft (366 m), and no mental decrement at 1,500 ft (458 m), and useful work may be done.

Decompression

Peculiar anomalies appear when a decompression schedule may be used successfully for the treatment of decompression sickness from 455 ft (138.8 m), but not for a dive to 300 ft (91.5 m). It is thought that exercise increases the likelihood of decompression sickness (Van Der Aue et al. 1949), so it is possible that an inactive patient may escape where others who remain active succumb.

The initial rate of 40 ft (12 m) per hour in the 1,500 ft (458 m) dive is obviously too fast and the approximately 20 ft (6 m) per hour rate used by Buhlmann et a!. (1970) would probably be safer. Assessment of all our experience suggests that for increasing depth a gradually reducing rate of exponential decompression is required and not a single rate. Future decompressions from these depths may best be begun with a slow linear bleed, changing to an exponential bleed and eventually, nearer the surface, changing again to a step decompression. This might prevent the bend of insidious onset persisting into a chronic state which resists conventional treatment in the way that Diver A's bend did.

At shallower depths labyrinthine and other serious forms of decompression sickness usually respond to rapid recompression (Barnard 1967), but in very deep dives the use of recompression appears to be limited by HPNS. If this is so the onus is even more than before on prevention of decompression sickness and, as permanent physical damage may result, a physician may feel bound to advise against exposures such as this in the future.

Future

This dive can only be described as a scientific experiment in a field where progress is rapid, and already a dive to 1,700 ft (518-5 m) has been conducted at the Experimental Hyperbaric Centre in Marseilles, using the compression technique evolved at RNPL and what appears to be a decompression schedule from the same stable.

REFERENCES

- Barnard E E P (1967) In: Proceedings ofThird Symposium on Underwater Physiology. Ed. C J Lambertsen. Baltimore;
- p 156 Buhlmann A A, Matthys H, Overrath G, Bennett P B, Elliott D H & Gray S P (1970) Aerospace Med. 41, 394
- Chouteau J, Jianco V, Oriol P, Coulboy R, Aquadro C F, Alinat J & Andrac C (1967) Ann. Anesthesiol. franc. 8, 238
- Royal Naval Physiological Laboratory (1970) Interim Report IR 1/70. Interim report of some physiological studies during 1,500 ft simulated dive (unpublished)
- Van Der Aue 0 E, Kellar R J & Brinton E S (1949) Experimental Diving Unit Report Number 1-49. United States Navy (unpublished)