Diving: occupation or physiological experiment?

Recent advances in deep diving physiology: the compression profile

Z Török MFOM *Environmental Sciences Division, ARE Alverstoke, Gosport, Hampshire PO12 2DU*

Keywords: saturation diving; compression

Papers read to Section of Occupational Medicine, 19 April 1988

Introduction

When a diver works under water, his body is subject to increased pressure, and his respirable gases must be supplied at the same pressure. During the period he spends at work, his body will absorb more and more gas until it is saturated about 2 days later at the new pressure of his environment. North Sea commercial divers, for example, typically spend around 3 weeks continuously 'in saturation' where in the habitat or on the job at, say 200 m depth, the pressure of 200 metres seawater (msw) is 21 times that of the atmospheric pressure, i.e. the pressure his body was 'designed' to function at.

The gas used is helium, containing about 0.4 bar oxygen. As the diver is returning to the surface, pressure must be decreased gradually to prevent the formation of gas bubbles in his body that would cause various forms of decompression sickness, some of which may be life-threatening. In the above example the saturation diver would need at least 8 days to be brought back safely to surface pressure. The time penalty for safe decompression is thus quite great.

To investigate changes to the diver's various physiological functions caused by the increased gas pressure is the task of institutions like the Admiralty Research Establishment (ARE) at its Alverstoke site. Equally important is the task of defining the limits of safe operational parameters to be used in various diving techniques and procedures. Implicit in all this is problem solving where possible. The first stage of such work, called a simulated dive, takes place in laboratory-based pressure chambers using volunteers who may or may not be qualified professional divers. All such research at Alverstoke is conducted in accordance with the Helsinki Declaration governing experiments with human subjects, and is scrutinized by an independent ethical committee.

Physiological effects of high pressure during diving fall naturally into 4 groups. Those due to the procedure of increasing the pressure in the initial phase of a dive are called compression or rate effects (positive dp/dt), and may subside during a period of time when the pressure is held constant. The second group (depth effects) are caused by the presence of high pressure or the inert gas used, typically nitrogen or helium. Thirdly, decompression effects are caused by decreasing pressure (dp/dt negative), an extreme example being decompression sickness, the 'bends'. All the above changes are reversible, this being not so in the case of effects in the fourth group, consisting of lasting damage. Too fast decompression is responsible for all the important effects here, for example ischaemic necrosis of bone or nervous tissue. The significance of this group of occupational injury is such that even the possibility of hidden yet sustained damage must constantly be monitored, since the potential is ever present[1].

Historically, physiological diving research was started in response to the unacceptable incidence of decompression sickness in early construction work where diving was essential[2]. Significant improvements were made by defining decompression tables, i.e. pressure-time profiles along which decompression may follow safely. Pain caused by too fast decompression was the most important criterion in these early experiments[3]. The present paper will concentrate on the compression phase. In a somewhat analogous process, compression profiles were designed to reduce the incidence of the high pressure nervous syndrome (HPNS). The criteria, in addition to the symptoms, were provided by tools of applied physiology.

The high pressure nervous syndrome (HPNS)

Exposing men to pressure which is increased too fast and too far initiates a variety of ill effects known collectively as HPNS[4,5]. Tremor, the first of its components to be noted, is accompanied by brisk myotatic reflexes[6], dysmetria and ataxia. When postural hand tremor is measured with a small accelerometer, the frequency spectrum shows its energy to be distributed approximately like that of normal physiological tremor, with a peak around 10 Hz. There may be in addition a smaller increase around 3-4 Hz. Increases in energy of about 8 times the subject's own normal is frequently seen in HPNS; increases over 20 times the normal value have been reported. Tremor of intentional hand movements during HPNS is seen to be roughly proportional to the degree of intended precision of the task. It will make its presence felt as a decrement in any performance test having an ingredient of manual dexterity, including handwriting. There is a noticeable diurnal variability - most tremor is shown usually first thing in the morning, least around midday.

Subjects learn quickly to modify their approach to tasks in order to cope with or to disguise their tremor and ataxia, the casual observer noting marked improvement within a day or two. Quantitative results using power spectral comparative techniques indicate that in spite of this an increased hand tremor of, say, 4-6 times the subject's own normal value, is likely to persist some days into decompression from a deep dive exceeding 300 msw or so. This group of disturbances of motor function is thus neither a rate effect nor a pressure effect, but a combination of both. Its transient component is larger than the steady-state portion; the latter, however, appears to be more refractory to changes of the compression profile in different simulated dives, attempting to reduce the HPNS.

0141-0768/89/020072-03/$02.00/0

The earliest sign of HPNS during compression is opsoclonus[5]. It is an involuntary, constant jitter of the eyes, random in direction, and 1-2 degrees of arc in magnitude. It is below the resolution of conventional electro-oculography, but is easily seen using magnifying goggles that illuminate the eyes depriving the subject of visual fixation (Barany spectacles). Opsoclonus is not seen shallower than 160 msw during compression, and then only if HPNS has been triggered and is imminent. It is probably the best early warning sign.

Another eye sign, gaze nystagmus, appears somewhat later, and is useful only in subjects in whom it is normally absent (about two-thirds of 20 or so dive subjects). It is less repeatable than opsoclonus. Care must be taken to ensure that the subject holds his gaze in a lateral position just within his field of binocular vision. Its appearance and severity (rate and whether sustained) may be regarded as an indicator of, in this case, environmental stress on the nervous system.

The vestibulo-ocular reflex is one of the compensatory eye movement mechanisms. It tends to aid the continued maintenance of gaze on a visual target during movement of the subject (or just his head), thereby aiding visual perception. Vision itself is not necessary for this reflex to operate; indeed, experiments to describe it quantitatively are best performed in total darkness, with the subject's eyes open. For example, if the subject is turned through an arc of 90 degrees to his right, whilst looking straight ahead at a picture on the wall, his vestibulo-ocular reflex will turn his eyes 90 degrees to his left, so that when he stopped turning, his eyes will still be on the picture, now on his left. The gain of the flex is 90/90=1, i.e. eye movement in degrees of arc, divided by body movement in degrees of arc (output over input). Twenty turns to each side were used in experiments of this nature during compression. In HPNS the reflex is shown to be disrupted[7]. Its gain may increase or decrease compared with the subject's own mean normal value. Possibly worse still, it may become asymmetrical, i.e. left turns would induce eye movements 12% greater than right turns performed at the same time. It is hardly surprising that the subject complains of a series of motion sickness-like symptoms, including nausea and vomiting, in these circumstances of intravestibular sensory conflict.

Hyperbaric electroencephalogram (EEG) changes have been well established and, with power spectral techniques, useful calculated indices may be obtained[8]. Slowing is the most conspicuous feature in deep simulated dives. Theta band power (4-8 Hz) may increase sharply, with the alpha band (8-13 Hz) showing diminished activity. The alpha index, relating the amount of alpha activity first appearing on eye closure to the amount present before, is seen to decrease in HPNS. Large increases in delta activity are sometimes seen, and in at least one well documented experimental dive this EEG feature was so disconcerting that further compression was abandoned for fear of cerebral cellular hypoxia[9]. In general, increased delta activity is not sufficiently repeatable to be a useful indicator. Its dependence on the subject's state of arousal may partly be responsible. Other hyperbaric EEG changes, such as those concerning the beta band (above 13 Hz), sleep patterns and paroxysmal waveforms, are similarly too variable to provide indicators for judging the limiting parameters of safe compression procedures. A peculiar problem of using the spontaneous EEG in this way is that the skill and efficiency of a diver may remain as high as ever in spite of altered brain function at depth, as evidenced by the above well-defined hyperbaric changes in his EEG[10]. Whereas unaltered physiological function of the brain is in itself desirable in an environmental exposure, one may well question the utility value of such criteria in themselves.

Psychological performance tests measuring psychomotor or essentially cognitive function have been frequently used in deep diving, as the results are of direct operational relevance. A 1984 review[11] considers them in the context of other ways of assessing the various compression profiles so far used in man. The reviewer concludes that a residual decrement of cognitive test performance of, say, 10% may be inevitable. The question is then asked whether this amount of functional loss in deep saturation divers is acceptable from the point of view of occupational health and hygiene.

Compression tables (profiles)

Using the criteria described in the previous section, experimental dives were conducted at ARE Alverstoke to differentiate compression rate effects from effects of increased pressure per se. To do this, a rate of compression was chosen which in practical terms can only be described as very slow. The compression profile consisted of a daily 'dose' of 60 msw (6 bar) increase of pressure in 6 increments of 10 msw each at 1 msw/min. Though the planned maximum depth of 420 msw was reached on day 7, the classical full-blown HPNS developed. Unlike in the prevention of decompression illness, where, in general, a slow decompression is a safe one, even extremely slow compression rates will not prevent HPNS. The compression profile has got to be right.

One such profile would reach 540 msw on day 5, provoking only minimal signs of abnormal nervous system function. Stages of 180, 300, 420, 420 and 540 msw were reached on consecutive days. The pressure was increased quickly at rates of up to 5 msw/min in two 60 msw steps, then held constant until the next day. Whilst this compression table is unlikely to be optimal, it is a practical one. Further increases in compression rate can be achieved by adding nitrogen or perhaps hydrogen to the diving gas mixture thereby increasing complexity, and introducing further problems. These 'tri-mix' procedures are not yet generally accepted in deep diving, and remain experimental.

References

1 McCallum RI, Shields TG, Minsaas B, Elliott DH (eds). *Long-term neurological consequences of deep diving.* Stavanger: European Undersea Biomedical Society, 1983

2 Bert P. *La pression barometrique.* Paris 1878 (Republished by Undersea Medical Society, Bethesda, 1978)

3 Boycott AE, Damant GCC, Haldane JS. Prevention of compressed air illness. *J Hyg London* 1908;8:342-443

4 Bennett PB. The high pressure nervous syndrome in man. In: Bennett PB, Elliott D, eds. *The physiology and medicine of diving and compressed air work.* London: Baillière Tindall, 1975

5 Torok Z. The symptomatology of HPNS - a proposed classification. In: Elliott DH, ed. *Eighth Annual Congress of the European Undersea Biomedical Society.* Lubeck: EUBS, 1982: 104-19

6 Harris DJ. Hyperbaric hyperreflexia: tendon-jerk and Hoffmann reflexes in man at 43 bar. *EEG Clin Neurophysiol* 1979;47:680-92

7 Hempleman HV, Florio JT, Garrard M, *et al.* UK deep diving trials. *Phil Trans R Soc Lond* 1984;**B304**: 119-41
8 Torok Z. Deep diving: trimix and heliox compared by EEG and tremor criteria. In: Bachrach A, Matzen M, eds. *Underwater Physiology VIII.* Bethesda: Undersea Medical Society, 1984: 697-705
9 Fructus P. Deep diving. In: *Proceedings of Conference "Oceanology International", Brighton.* London: Society for Underwater Technology, 1969
10 Rostain JC, Lemaire C, Gardette-Chauffour MC, *et al.* Criteria analysis of selection for deep diving. In: Bachrach A, Matzen M, eds. *Underwater physiology VII.* Bethesda: Undersea Medical Society, 1981: 435-43
11 Torok Z. Behaviour and performance in deep experimental diving. In: Bachrach A, Matzen M, eds. *Underwater physiology VIII.* Bethesda: Undersea Medical Society, 1984: 739-60

(Accepted 7 June 1988)

Treatment of compressed-air decompression accidents

N K I McIver MB MFOM *North Sea Medical Centre, Gorleston on Sea, Norfolk NR31 6SG*

Keywords: decompression sickness; barotrauma; divers; tunnellers

History

Following Sir Robert Boyle's observation of bubbles in live tissues in a famous exposure of a viper to a vacuum decompression in 1670, decompression illnesses in man were first recognized in the 19th century in tunnellers and miners.

In 1841, Triger[1] first described decompression illness in coal miners in pressurized shafts as caisson disease when large capacity compressors were developed to pressurize bridge building foundations to prevent flooding and numbers of men were exposed to raised pressures for prolonged periods.

In 1854, Pol and Watelle described the relation between symptoms and depth and duration of exposure, and speed of decompression[2]. In 1847, they also observed relief of symptoms by increasing ambient pressure and suggested gas bubbles might be the cause of the problem.

In 1872, Friedburg noted the similarity between severe decompression sickness and surgically induced arterial gas embolism, and Gal described the first case of neurological decompression sickness in a commercial diver (Elliott 1985, personal communication). Practical deep sea diving commenced in 1819 when Augustus Siebe designed the first diving dress. In 1878, Bert[3] analysed the blood gases and bubbles which formed in experimental animals with decompression sickness and identified nitrogen as the principal cause. He recommended recompression and elevated partial pressures of oxygen as therapy for decompression sickness. No procedure for standard decompression was developed until Heller, Mager and von Schrötter proposed, in 1900, a linear decompression rate of 20 min for each 33 ft (1 atmosphere absolute) which was successful for dives down to 160 feet of seawater.

Haldane was appointed by the Admiralty in 1905 to develop safe decompression procedures. They were published in 1908[4]. Subsequent air decompression tables for the next 50 years were based on these decompression procedures and later modified for divers[5]. The hazards of air diving without safe decompression were shown by Blick[6] who described 200 cases of decompression sickness, of whom 60 died in 1909.

Tunnellers had their own decompression tables and therapeutic procedures[7]. They now follow therapeutic procedures according to the Construction Industry's CIRIA code of practice (1982).

Decompression sickness

Following the earliest descriptions of decompression sickness (DCS) in 1872, there was a report of cases of 'divers' paralysis' in pearl fishers. Cases of slight paralysis were common after removal of diving dress but 'generally recovering completely'[8].

In tunnelling, the experience of the first Dartford Tunnel was recorded[9] which describes 685 cases of decompression sickness, 650 (94.9%) of which were 'simple Type 1 bends' and 35 (5.1%) having symptoms other than pain described as 'serious Type 2 symptoms'. All cases were successfully treated without permanent disability using minimum pressure required for relief of symptoms followed by slow decompression with occasional 'soaks' by holding at various depths during ascent. This method was considered more satisfactory than trying to force the bubbles wholly back into solution by use of high pressures.

The US Navy reviewed 20 years of experience of treating cases of DCS in 1964[10] and found an overwhelming preponderance of Type I as opposed to Type II DCS. This has now been entirely reversed with more serious symptoms being more common[11,12]. The apparently mild sensory symptoms may herald onset of spinal or cerebral DCS in sports divers[13]. This has been borne out over the last 23 years at this Centre where of a total of 406 cases recorded there were 80 Type 1 cases as opposed to 178 Type 2 cases. (The remainder were 54 cases of barotrauma from all causes and 94 cases subsequently proved to have 'non-dysbaric' illness).

Approach to therapy

The basis of therapy continues to be recompression, an elevated partial pressure of oxygen and use of certain drugs. Recompression reduces the gas volume (Boyle's Law) and tends to drive the gas back into solution (Henry's Law); if applied soon enough it should restore circulation. Addition of elevated oxygen partial pressure will increase inert gas washout, reduce tissue hypoxia and possibly oedema, and improve oxygenation.

0141-0768/89/020074-06/$02.00/0