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U.K. Deep Diving Trials [and Discussion]

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U.K. deep diving trials

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Using a breathing medium of 40 kPa oxygen, remainder helium, 18 volunteer subjects participated in a series of 15 exposures to pressures equivalent to depths of 180–540 m s.w. The time of exposure at these pressures was mostly 2 days, except for the 540 m s.w. exposure, when 6 days were spent at full pressure. Compression procedures, based upon placing 'stages' at 60 m s.w. intervals, evolved with experience and proved to be a highly successful way of achieving acceptable pressure–time courses. Decompression combined slow linear release of pressure with overnight halts for sleep. On one occasion a depth of 660 m s.w. was reached by breathing 40 kPa oxygen, 10% nitrogen, remainder helium. Throughout all exposures, teams of investigators followed the changes in cardiovascular, respiratory, haematological, neurophysiological and metabolic status, and mental performance of the volunteers. Some major findings were that the neurophysiological and behavioural changes could be assigned to the motor, or vestibular, or cerebral, or autonomic systems, and were mainly first observed during compression. The subjects suffered, apparently from severe nitrogen narcosis, when breathing 10% (by volume) nitrogen in oxygen–helium at 420 m s.w. Lung ventilation was remarkably adaptable to the oxygen requirements of exercise at all depths, but cardiac output was adversely affected at 540 m s.w., particularly for heavier workloads. Ventilatory responses to carbon dioxide were significantly elevated after diving. Thermal balance was seen to be precarious, but nevertheless it was achieved by the normal subjective assessments of comfort. Water loss was affected by diminished evaporation from the skin. Skin temperature sensitivity was changed and took many days after the dives to return to normal. Energy requirements increased for work purposes, but basal metabolic rate was undisturbed. Body chemistry altered at pressures in excess of 300 m s.w., for example thyroid hormone and nitrogen balances were affected. No decompression sickness was encountered until the pressures were low, but marked haematological changes could occur during decompression. Every change that occurred during these dives reverted to normal, mostly before the end of the decompression. It is concluded that diving with oxygen–helium breathing mixtures to depths as great as 540 m s.w. can be effective and safe. An attempt is made to assess the physiological significance of the principal findings.

Introduction

When the present series of hyperbaric experiments began at the Admiralty Marine Technology Establishment (Physiological Laboratory) the aim was to define the limits of usefulness of helium as a diving gas. To pursue that aim the Laboratory possessed a small two-compartment pressure chamber (1.68 m internal diameter and 4.24 m internal length) with a maximum working pressure of 7 MPa. It was decided to expose the human volunteers in pairs, and a versatile life-support system was constructed to fulfil the demands of the Ethical

Committee and the investigators. With two men expected to be living for many days in the somewhat cramped conditions of this pressure chamber it was necessary to see whether modification of any of the proposed investigations would be required, and also to ascertain if the volunteers were able to maintain healthy mental and physical states.

Accordingly, the first simulated dive was a 9 d exposure to a pressure equivalent to a sea-water depth of 3 m with air as the breathing gas. The next two dives were also at 3 m s.w. for 9 d, but the breathing gas was now 30 kPa oxygen, remainder helium. These three control experiments demonstrated that men could live in the chamber for quite prolonged periods without exhibiting any signs of stress. After this important and reassuring point had been established the first of the deep dives was made. From the outset it was decided that the breathing mixture would always possess a partial pressure of 40 kPa oxygen, except for the very final stages of the decompression, when fire hazards dictated a lower value. The nitrogen partial pressure in the chamber was reduced to as low a value as practicable, by gas-rinsing procedures, and a day spent at 3 m s.w. while breathing the oxygen–helium mixture. This period at low pressure was used to establish control values for the numerous tests to be attempted at depth later. Compression occurred at 1 m s.w./min to a maximum depth of 200 m s.w., which was then held constant for a further 8 d before decompression commenced. This dive was entirely uneventful subjectively, and objectively only minor, or expected, changes (thermal exchange, respiratory resistance etc.) were seen. This also served as a control exposure, and provided the first thorough test for the special hyperbaric techniques developed by the several teams investigating various aspects of neurophysiology, psychology, respiratory physiology, cardiovascular physiology, energy exchange, dietary requirements, haematology and decompression sickness. Their findings will be outlined later, but what will now be given, in chronological order, is a description of the pressure–time courses of the experiments upon which their findings are based, and the reasoning behind the way the dive programme progressed.

Dive 5 was planned to follow the same general pattern as the 200 m s.w. dive, with a control period on oxygen–helium at 3 m s.w. followed by compression to depth at 1 m s.w./min. The intention was to reach 300 m s.w. at this compression rate, but in the event a depth of only 240 m s.w. was attained when nausea, dizziness, and vomiting, present in both the volunteers, enforced a halt. Some improvement occurred after approximately 2 h and compression was recommenced. The planned maximum depth of 300 m s.w. was reached, but both men could best be described as miserable for the next 2 d. All experiments were completed and very little change could be seen from analysis of their behavioural responses after the dive. Nevertheless these men were not in a desirable state for practical underwater work purposes for at least the first two of the 8 d they spent at this depth.

It was thought possible that the nitrogen-free period in the pressure chamber before compression could be removing protective effects claimed for that gas, and consequently these sickness problems were worse than they need be. This possibility was tested on the next dive. Compression began with no attempt to remove nitrogen from body tissues. However, near the same pressure level as on the previous dive, the 1 m s.w./min rate of compression had to be abandoned because of severe distress in both volunteers. This time a prolonged 1 d halt at a constant depth of 240 m s.w. markedly improved their condition and 300 m s.w. was reached by resuming the 1 m s.w./min rate of compression.

The divers took about 2 d for complete recovery from the ill effects induced by the compression procedure. This was taken as demonstrating that the previously used control

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periods had had little or no influence on the ill effects provoked by compressing beyond 200 m s.w. at a constant rate of 1 m s.w./min.

As the principal aim of these experiments was to define the limits of usefulness of helium it was quite clear that we were becoming involved in establishing compression profiles before knowing which effects were due to pressure alone, and which due to the rate of change of pressure. It was therefore decided to compress at an extremely slow rate of compression and thus eliminate this variable from future results. A rate of 60 m s.w./d was chosen as representing a sufficiently slow rate to satisfy this requirement, and was achieved during Dive 7 by placing stages of 2 h duration at 10 m s.w. pressure intervals, followed by a 12 h overnight rest period (see figure 1). After 4 d, a depth of 240 m s.w. had been attained with both divers completely free of symptoms. However, upon the completion of the first 10 m stage of the fifth day one of the divers complained of a sore throat, and it was decided to halt the compression to see whether the sore throat was a serious threat, or merely a passing problem. Two days were spent observing the diver before it was concluded that the sore throat had returned to a normal state and permission given by the doctor for resumption of the experiment. Compression continued at 60 m s.w./d until a depth of 420 m s.w. was reached. This depth was held for 2 d before the start of decompression. The men were remarkably unaffected by this particular compression profile, although there were occasional outbursts of hand tremor and some very infrequent myoclonic jerkings as a reminder that slight abnormalities were present, but they were undoubtedly extremely fit both mentally and physically. A conclusion drawn from this dive was that a depth of 420 m s.w. could be reached, by using an oxygen–helium breathing mixture, with only the most trivial manifestations of the high pressure neurological syndrome (h.p.n.s.). The next dive was therefore a repeat of this dive, but without the enforced halt for the sore throat (see figure 1). A depth of 360 m s.w. was reached without any complaints and it seemed as though this was to be as equally trouble-free as the previous 420 m s.w. However, the final day's compression to 420 m s.w. produced a number of signs and symptoms that were far from trivial. The neurologist, who was following the oculo-vestibular reflexes at the time, stated that signs of abnormality began at about 390 m s.w. The volunteers were clearly very unhappy during most of the time at maximum depth. Nevertheless they were able to do all the tasks asked of them. For example, they successfully completed all venepunctures, worked hard on the ergometer, and even managed to pursue the dietary experiments, despite feeling wretchedly nauseous.

This result came as a complete surprise, because some 10 years previously an exploratory oxygen–helium dive to a maximum depth of 467 m s.w. had been completed at this Laboratory without any signs of distress in the subjects throughout the compression phase or the time at maximum depth (Bennett & Towse 1971), but the compression time to the depth of 420 m s.w. had only taken approximately 2 d. It was temporarily concluded that either individual variations of response to these environments were involved or that large pressure changes followed by prolonged (24 h) stoppages gave a 'provoke–adapt' cycle of events that was more effective than gradual change.

Whatever the explanation it was clearly necessary to repeat these earlier results and so the next dive, 9 B, was essentially a repeat performance of this early pressure–time course with extra stages added to reach a depth of 540 m s.w.

Two professional divers acted as volunteer subjects and they reached 420 m s.w. in 2 d compression time with almost no signs or symptoms of compression effects. This gave a total

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of 4 volunteers who had completed this relatively rapid time course without problems, and it was becoming less likely that individual variation had been responsible for the early dive success. After 20 h at 420 m s.w., the 2 men were rapidly compressed to 540 m s.w., with a 4 h stop at 480 m s.w., which both men reached with only minimal signs of compression effects. However, after 3 or 4 h at 540 m s.w. their condition had changed considerably and they had developed all the signs and symptoms encountered by the Dive 8 men, i.e. nausea, vomiting, gaze nystagmus etc. Naturally this tended to affect the whole experimental programme, but nevertheless all the physiological investigations were successfully completed during this period.

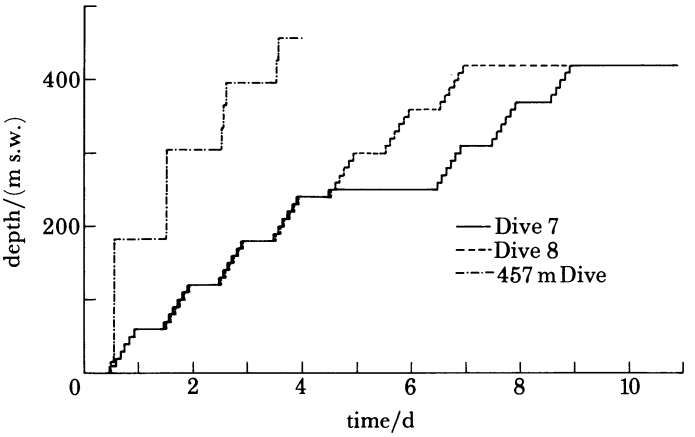

FIGURE 1. Comparison of the compression and time at depth for Dives 7 and 8 (420 m s.w.) and the early 457 m s.w. (1500 ft s.w.) dive.

Just before this experiment the U.S. Navy had placed 6 volunteers at a depth of 540 m s.w. (1800 ft s.w.) and despite a very cautious compression procedure had experienced considerable compression sickness difficulties (Spaur 1979). These observations, combined with the Dive 9B results, gave the impression that at 540 m s.w. the body had reached the limits of adaptability to raised partial pressures of helium.

The next dive, 12B (see figure 2), employed a breathing mixture composed of 40 kPa oxygen, 10% nitrogen (by volume) remainder helium. The nitrogen partial pressure of such a breathing gas increases with increasing pressure and, for example, at 420 m s.w. would be 430 kPa. The 7 h compression profile used as far as 420 m s.w. was exactly that of ATLANTIS II (Bennett 1982). The 2 volunteers arrived at this depth suffering from what appeared to be severe nitrogen narcosis, i.e. giggling and irresponsible behaviour that prevented the investigators from making their experiments. This was the first time such a loss of experimental evidence had occurred in a compression. Both men were equally affected but after a 2 d halt at 420 m s.w. both had changed to much more responsible behaviour, which permitted collection of experimental data. They were next compressed to 540 m s.w., where their general behaviour seemed to be the same as on leaving 420 m s.w. They stayed overnight at 540 m s.w. before being further compressed to 660 m s.w. This final depth was reached with both men euphoric and clearly afflicted by a less serious form of the nitrogen narcosis than had appeared on arrival at 420 m s.w. Nevertheless the experimental programme went ahead at 660 m s.w. After 4 h there was a marked change in behaviour when both men became extremely fatigued at the slightest exercise. Their general condition, in addition to this extreme tiredness, alternated between the euphoria and irresponsible behaviour associated with nitrogen narcosis and the nausea,

vomiting etc. of compression sickness. It was expected that an improvement might occur upon staying at 660 m s.w., but after 37 h this did not happen and decompression was begun. Once again, data collection had been severely disrupted by the inability of the volunteers to cooperate with the investigators.

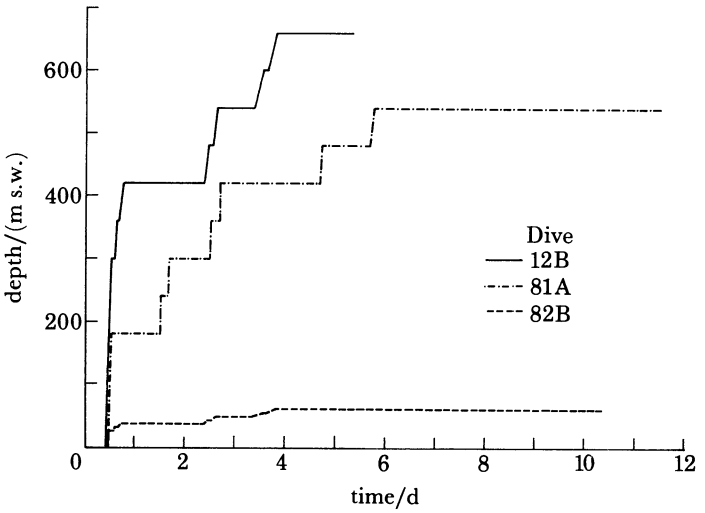

FIGURE 2. Comparison of the compression and time at depth for Dives 12B (660 m s.w.), 81A (540 m s.w.) and 82B (61 m s.w.).

Experience from the 12B dive and accumulated evidence from elsewhere (Rostain *et al.* 1977), with breathing mixtures of nitrogen, helium and oxygen, showed that much more compression time would probably be needed to minimize the narcosis effects. So from a practical diving standpoint it seemed that no major advantages, but several disadvantages, accrued from the use of these ternary gas mixtures.

It was therefore decided to re-investigate the 540 m s.w. depth (Dive 81A) by using oxygen–helium, but instead of proceeding as before, from 420 to 540 m s.w. in a single pressure change, to spend much more time at the intermediate depth of 480 m s.w. and allow further adaptation. Fortunately the same 2 volunteers participated in this experiment and therefore the comparisons between these last 2 dives are better founded than usual. The pressure–time course can be seen in figure 2 and as far as 420 m s.w. is identical to the previous 9B attempt at 540 m s.w., which in turn was closely similar to the much earlier 457 m s.w. dive. Once again both volunteers arrived at 420 m s.w. in excellent physical and mental condition and this makes a total of 6 different men who have satisfactorily reached this depth by using the 2 d compression procedure, thus demonstrating the practical effectiveness of staged compression profiles. The 2 men, after a 2 d halt, were next compressed to 480 m s.w. where no further changes were noted, and following a 1 d halt at 480 m s.w. proceeded to the maximum depth of 540 m s.w. where they remained for 6 d before decompression was instituted. During the whole of this time at maximum pressure they behaved very responsibly and all experiments were completed properly. They ate well and showed no indications of deterioration throughout the quite prolonged sojourn at 540 m s.w. Some tremor of intentional movement was present; in fact the same levels as those found on the 10% (by volume) nitrogen mixture of Dive 12B at this same depth.

This final deep dive of the series yielded the practical finding that effective diving on oxygen–helium mixtures is clearly possible if a suitable compression procedure is used. From the basic physiological standpoint it became necessary to examine why the subjects had been so severely narcotized on compression to 420 m s.w. in Dive 12B with a partial pressure of only 430 kPa nitrogen. So an oxygen–nitrogen dive was undertaken, with 2 volunteers, to a final depth of 61 m s.w. This dive reproduced, at all times, the exact equivalent partial pressure of nitrogen to those encountered during Dive 12B (see figure 2). With compression at these slow rates very little impairment of performance was detected (Logie & Baddeley 1983) at any time throughout the whole exposure. The conclusion they reach is 'This suggests that the large decrements found with Trimix were not simply due to the partial pressure of nitrogen present'.

The following sections give brief accounts of the principal physiological findings obtained during the course of these experimental dives. These accounts will be followed by a discussion that will attempt a 'global' view of the various investigative areas and provide some practical and theoretical conclusions.

Thermal comfort

The oxygen–helium diving environment is typified by the need for fine and precise control of the gas environment at an elevated temperature. On exposure to the helium environment, even at a shallow depth, the increase in thermal conductivity and specific heat (relative to air) require a higher temperature for comfort. Increases in density require a further elevation of temperature and only minor fluctuations (under ± 1 °C) can cause discomfort. Some examples of the relation between depth and comfort temperature are shown in table 1.

Table 1. Relation between depth and comfort temperature

depth/(m s.w.)	60	120	180	200	240	300	360	420	540
mean comfort temperature/°C	28.8	30.5	30.9	30.9	30.9	31.1	31.6	31.3	31.7
daily variation/°C	±0.8	±0.3	±0.5	±0.6	±0.4	±0.5	±0.7	±0.7	±0.4

The changing importance of each avenue of heat loss (convection, evaporation and radiation) with increase in density can be examined by using the classical means of partitional calorimetry. The results of such practical experimentation (Hayes 1977) bore out the findings of earlier theoretical studies (Flynn *et al.* 1974) and showed that convection, both respiratory and skin, becomes a major pathway at the expense of other modes of heat transfer. An additional factor to consider is the important role of respiratory convective heat loss; in the overall thermal balance respiratory losses can become a major threat to the diver working hard in cold water. He may lose more heat during the inspiration–expiration cycle than is generated by metabolism.

At rest the man living in a comfortable air (100 kPa) environment is likely to lose 10% of his metabolic heat production during respiration, 40% from surface radiation, 25% via convection and a further 25% due to evaporation. At 300 m s.w., convection accounts for 60% of total heat loss, 20% is due to respiration and only 15 and 5% for radiation and evaporation respectively. As the man is close to the surrounding chamber walls the temperature of the steel can cause marked changes in his radiation heat loss. The comfort of the man can be

accomplished by fine adjustment of both gas temperature (convection loss) and wall temperature (radiation loss). In practice it has been found that the best means of attaining comfort and good environmental control is to provide chamber wall heating at the average desired comfort level and to make fine adjustments for different levels of activity by controlling the gas temperature. The other advantage of wall heating is that it provides a large thermal inertia to resist rapid changes in gas environmental temperature occurring from rapid gas movement, as in compression. It is recommended that both gas and chamber wall heating are used to provide optimum environmental control.

The marked reduction in evaporative heat loss is due both to the increase in density and the high level of humidity often found. An increase in density reduces the mass transfer rate of water vapour diffusion from the skin surface and gives rise to the perceived sensation of skin wetness. The reduction in mass transfer can be theoretically derived from the work of Chilton & Colburn (1934) and was found to agree well with empirical findings (Carlyle *et al.* 1979 *b*). There is an increase in total body water excreted at depth, with the diuresis augmented at the greatest density (Dives 7 and 8). This appears to be a compensatory reaction for the lack of water that is lost from insensible perspiration when fluid intake is maintained. However, when the humidity is lowered in the chamber to allow for a rate of transfer commensurate with normobaric living, then the diuresis is not found (as in Dive 9B). Mean insensible losses in air (100 kPa) were measured in an environment of approximately 12 mg (H_2O)/l and were found to be about 27 g/h. At 350–420 m s.w. on Dives 7 and 8 the average insensible loss was only 3 g/h, but would be returned to the 100 kPa level by a reduction in hyperbaric environmental humidity to less than 2.5–3.2 mg/l (Dive 9B).

There were reports in the literature that equated exposure to oxygen-helium with dramatic loss of body weight and a stimulation of thermogenesis as a consequence of a sustained thermal drain. The possibility was raised of a stimulus to metabolism in spite of a supposedly comfortable condition and it was argued that a man may not be capable of correctly assessing his own temperature for obtaining thermal balance. Evidence stated for this hypothesis were the large weight losses incurred by a number of divers in spite of a supposedly adequate energy intake (Webb 1976), high resting values of oxygen uptake (Raymond *et al.* 1968) and elevated resting rectal temperature (Pilmanis *et al.* 1975). Weight loss due to an excessive energy expenditure was complicated by the observed negative water balance in a number of dives as well as by vomiting during compression.

One possible approach to an analysis of the problem, and one adopted in this series of dives was to embark on a complete examination of the energy balance of the diver during the total time at raised pressure and for a time before and after the dive (see below). Another approach used was to measure the ability of the man to perceive a given temperature and note any change that may occur during the saturation diving exposure. A convenient index of sensation is the threshold value of perception, and by using the psychophysical approach it is possible to analyse perception on a quantitative basis. The recorded sensory observation was the first indication of a thermal transient occurring as the temperature moved away from a previously adapted level of 22–39 °C. The temperature transient was imparted to the skin by using a Peltier module (625 mm²) applied to the surface of the lower arm (Hayes 1979).

The skin was cooled from the adapted level (x) at a constant rate to the first recognizable threshold level (y). In 100 kPa air the regression $y = 0.82 + 0.89x$ ($r^2 = 0.99$) describes the relation between threshold and starting temperature. As the dives progressed the slope of

the relation gradually fell, and by the third week at pressure became $y = 5.57 + 0.74x$ ($r^2 = 0.99$). The change in threshold is particularly noticeable within the range $x = 30$–39 °C ($p < 0.05$). Upon return to 100 kPa there was a gradual return to the response before the dive, with levels only marginally different after 2 weeks ($0.05 < p < 0.1$) and complete after about a month. Based on an analysis of receptor function proposed by Kenshalo (1970), a plausible statement is that prolonged hyperbaric oxygen–helium exposure may alter the temporal course of short-term temperature adaptations. The site of this change in perception is uncertain. It could be central and interpretive rather than owing to any change in the receptor itself. Alternatively, any fluctuations in the subcutaneous blood flow could give rise to the measured differences because the heat transferred could be noticeably increased by greater peripheral perfusion. It seems unlikely that these minor changes in sensitivity cause any marked alteration in thermoregulation function. However, it is worth noting that the effect is exaggerated with time in oxygen–helium and this may have some impact on much longer exposures.

Metabolic balance

A known fixed diet was fed to the subjects on each of the dives and this provided a stable base line from which changes in control values, measured before the dive, could be observed free from diet errors of both a chemical and thermogenic nature. Although there was no change in basal metabolic rate down to 540 m s.w., there were increases in energy expenditure in the h.p.n.s. zone during light exercise, including postural support (Carlyle *et al.* 1980). This resulted in an overall negative energy balance that was exaggerated by fixing the diet to the normal surface requirement.

Negative nitrogen balances in this series of dives have been consistently recorded in divers deeper than 350 m s.w. (Garrard *et al.* 1980; Carlyle *et al.* 1978). Negative potassium balances have also been recorded at the same time, which confirm a loss of lean body mass during the period of the negative nitrogen balance. Positive balance is encountered before the end of the dive, which indicates that muscle wasting due to inactivity is not a contributory factor. However, the stimulation of protein catabolism is not generalized because there is evidence from 24 h urinary hydroxyproline and serum proline iminopeptidase levels to suggest that collagen metabolism and turnover is stable (Carlyle 1978; Carlyle *et al.* 1979). The disturbance to protein metabolism is nevertheless significant and supportive data from serum amino acid studies, skeletal muscle enzymology and liver function tests have demonstrated that the degree of dysfunction is sustained by pressure and is unrelated to the presence or severity of h.p.n.s. in an individual. A close link has also been demonstrated between alterations in protein metabolism and thyroid hormone levels in these divers.

During the deeper phases of each dive, increases in thyroxine and reverse tri-iodothyronine accompanied by no change in either tri-iodothyronine, tri-iodothyronine resin uptake or thyroid stimulating hormone have been observed (Andersen *et al.* 1982), and this occurs in men subjected to both deep Trimix and oxygen–helium dives irrespective of the presence of h.p.n.s. or narcosis. The phenomenon is pressure-related, as is the stimulation to protein catabolism, and occurs deeper than approximately 300 m s.w. Andersen *et al.* (1982) have suggested that the peripheral metabolism (de-iodination) of thyroxine is disturbed rather than the central stimulation of the thyroid. Similar alterations have been observed in certain clinical conditions (Henneman *et al.* 1979) but have not been previously reported in apparently healthy

subjects. Furthermore, the raised thyroxine levels above the upper limits of normal levels, accompanied by progressive decreases in creatine kinase and progressive increases in adenylate kinase, have demonstrated a remarkable analogy with clinical hyperthyroidism (Doran & Wilkinson 1971).

No evidence has been found for a gross disturbance in bone metabolism during these dives. Not only was collagen metabolism normal (Carlyle *et al.* 1979*a*), but also there were no significant changes in calcium and phosphorus balance or excretion rates.

A number of changes from circulatory hormonal levels measured before diving have been observed and reported (Carlyle *et al.* 1978*b*; Garrard & Carlyle 1982) and although these contribute to both the compression and decompression sickness phenomena, little threat to diver safety or performance can be inferred at this stage.

The evidence that has been accumulated clearly shows that the major metabolic disturbance by the hyperbaric environment at depths in excess of 300 m s.w. is a stimulation of catabolic metabolism. The divers appear to be in a condition similar to patients recovering from abdominal or orthopaedic surgery and also to subjects exercizing where there is a positive stimulation of protein catabolism accompanied by a depression of protein synthesis. It is possible that the hyperbaric environment stimulates the metabolic pathways concerned in these mechanisms. It is also likely that thyroxine plays a major role in these events.

There is no evidence to suggest that overall energy balance cannot be maintained, but the evidence is less clear for nitrogen balance. During Dive 81A both divers increased their protein intake by 80% at 540 m s.w. in the middle of the negative nitrogen balance period. One diver returned to positive balance while the other diver remained in negative balance with an increase in protein catabolism to cater for the extra intake. It is far less certain therefore that by raising the protein intake, stimulation to protein metabolism will be countered. Furthermore, it is difficult at this stage to predict how the disturbances in thyroid hormone chemistry may be accommodated.

Neurophysiology

Studies of the nervous system were conducted with two sets of objectives in mind. The analytical–diagnostic interpretation of data attempted to delineate parts of the nervous system responsible for abnormal functions observed. Secondly, by the use of objective and quantitative criteria wherever possible, the h.p.n.s. was used to assess the relative merits of various schemes of compression and of nitrogen added to breathing gas. There was considerable methodological overlap between the two studies.

Electro-oculography and direct observation demonstrated abnormal features of eye movements (opsoclonus, dysmetria and rebound) probably arising in the cerebellum or midbrain. Break-up of smooth pursuit pattern in following a visual target may be of midbrain or cerebellar origin. These and other related pathological signs typically appeared on compression at around 300 m s.w., and persisted up to the third day of decompression. Opsoclonus (small amplitude random ocular tremor) was the first positive sign to appear, often before 200 m s.w. was reached (Török 1981). It was usually followed by increased intention tremor and ataxia, which again suggests the cerebellum.

Another group of symptoms and signs composing the h.p.n.s. (Török 1982) is reminiscent of motion sickness: disturbed balance and vertigo, frequently of intermittent, sliding character

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nitrogen–oxygen alone to 61 m s.w. (Nitrox). The speed of increase of nitrogen partial pressure was identical in the Trimix and Nitrox exposures, but the speed of increase of helium partial pressure was greater in the Trimix dive than in the Heliox dive. Care was taken to minimize the effects of as many other variables as possible.

It has long been known that the e.e.g. of subjects suffering from h.p.n.s. is often modified, and some of these changes, for example, an increase in the 4–8 Hz (θ) band or decrease in the 8–13 Hz (α) band of the power spectrum, are amenable to quantitative analysis (Bennett & Towse 1971). Another h.p.n.s. component, namely increased postural hand tremor, may also be used as an index of h.p.n.s.

The ratio of power spectral estimates when summed for the 4–8 and 8–13 Hz band (the θ to α ratio) increased, by 3–8 times the subjects' own control values in both the Trimix and Heliox exposures about equally, and did not increase in Nitrox (Török 1983). Similarly, the summed power of hand tremor over 1–20 Hz increased almost threefold in both exposures, without changing in Nitrox.

The triangular design of this experiment permits the following conclusions. First, high hydrostatic pressure (or helium) present in Trimix and Heliox was accompanied by statistically significant increases in tremor and 4–8 Hz power of e.e.g. Second, nitrogen at partial pressures up to 0.56 MPa, high enough to modify behaviour in the sense of 'nitrogen narcosis', did not cause significant changes in either tremor or e.e.g. Third, nitrogen at approximately the same partial pressure did not decrease the magnitude of changes seen in the Heliox exposure.

So within the limitations of this study (four subjects, three exposures, slower compression in Heliox, data taken at equivalent points of pressure and different subjects in Nitrox) there is a clear result. Its interpretation is that until further work with Trimix is done, especially related to the alignment of the nitrogen dose with the compression profile, the addition of nitrogen in deep dives at present seems to offer little to offset its inherent disadvantages.

Cardiorespiratory physiology

The main aims of the exercise studies done during this series of dives were to investigate the link between CO_2 retention and raised ambient pressure, and to attempt to determine any ventilatory and cardiovascular limitations to exercise. To this end the following physiological parameters were measured: airway resistance, flow resistive work of breathing and compliance, maximum voluntary ventilation, total expired ventilation per minute, the gas exchange ratio, the rate of isometric inspiratory pressure development (dp/dt (max)), pulmonary capillary blood flow, pulmonary tissue volume, heart rate and breath by breath expired gas concentrations at the mouth.

As expected with increasing pressure, maximum voluntary ventilation fell as approximately the square root of the density. No consistent effect of pressure or gas composition was observed (Winsborough *et al.* 1980). Airway resistance and flow resistive work of breathing both increased with increasing gas density in a linear fashion, except in one subject who displayed a disproportionate increase in airway resistance at 660 m s.w. Compliance fell with increasing pressure but independently of gas density; this will be mentioned later in conjunction with changes to pulmonary tissue volume. Bearing these changes in mind, it is surprising that the ventilatory exchange ratio was well maintained at all depths and exercise levels except where ventilation approached maximum voluntary ventilation at that depth. However, dp/dt (max)

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was increased at 180 and 250 m s.w. sufficiently to maintain or enhance the ventilatory response to CO_2. The work of Linnarson & Hesser (1978) and Lopata *et al.* (1980) offer explanations involving reflex stimulation of the respiratory centres and enhanced diaphragmatic and respiratory muscle activity for a given normal drive. The ventilatory response at pressure was itself related to the response at the surface; it was maintained at pressure in subjects with a poor surface response, but fell at pressure in those with a brisk surface response.

The ventilatory response to CO_2 was significantly elevated after dives, the degree of enhancement being inversely proportional to the response before diving, but was not increased by repeated exposure to pressure. These effects appear to be independent of the gas mixtures used during the dives, and are not an artefact of the re-breathing procedure employed, since similar changes were seen in the steady state ventilatory response to CO_2. There was no consistent relationship between the observed changes and breathing pattern, and they seem not to be related to changes in lung volume or mechanics because airway resistance, compliance, and flow resistive work of breathing, were essentially unaltered after diving. It is also unlikely that the effects were due to exogenous CO_2, because the inspired CO_2 partial pressure was of the order of 0.2 kPa throughout the dives. Finally, since the ratio of total expired ventilation per minute to O_2 uptake per minute was well maintained at pressure, these findings are unlikely to be explained in terms of raised alveolar CO_2 partial pressures due to hypoventilation.

The cardiovascular data reinforces the earlier conclusion that CO_2 was not retained except at exercise levels approaching maximum voluntary ventilation at the highest gas densities. The limiting condition of the solution of the instantaneous CO_2 Fick equation used to analyse the data obtained from these dives (Winsborough *et al.* 1980) is that the pathways for the release of CO_2 should be infinite. The integrity of the model was not challenged on Dives 1–8, despite a consistent fall in exercise pulmonary capillary blood flow. There appears to be no limitation to the release of CO_2 under exercise conditions down to 420 m s.w. unless either effort independent flow limitation of ventilation is attained or a restrictive limitation to ventilation is imposed. The first 540 m s.w. Heliox dive resulted in a significant fall in pulmonary capillary blood flow, which could have been produced by either a perfusive or diffusive barrier to gas exchange. Differential analysis of the components of the solution for the flow suggested an increased rate of exchange between the pulmonary parenchyma and the alveolar space, but a decreased rate of exchange with the pulmonary capillaries for CO_2. Tissue carbonic anhydrase activity was therefore unaffected by pressure.

The decreased wash-out of CO_2 from the pulmonary capillaries could have been the result of an increased capillary–parenchymal resistance or a fall in pulmonary capillary blood flow. The possibility of a reduction in red cell carbonic anhydrase activity was identified as a preferential decrease in the rate of rise of the wash-in of CO_2 into the alveoli from the capillaries, when simultaneously compared to the rate of wash-in of N_2O into the pulmonary circulation (Winsborough 1980).

During the 660 m s.w. Trimix dive, therefore, a method of estimating the pulmonary capillary blood flow, which was not gas flow limited, was used simultaneously. The combined data supported the view that there was a significant reduction in pulmonary capillary blood flow *per se* at 540 m s.w., the effect being most evident for heavy work (Winsborough *et al.* 1981). This has since gained support from a further 540 m s.w. dive showing the same cardiovascular decrement with exercise. Both heart rate and stroke volume are decreased during heavy work at these pressures. The decrease in stroke volume is especially important and implies a decrease

in myocardial contractility. Associated with this is a fall in compliance, probably as a result of the increase in pulmonary tissue volume and the increase in respiratory work at increased gas densities, but pulmonary capillary blood flow does not appear to respond in the normal way to the work stimulus at these pressures.

There is also some evidence that there may be an increasing resistance with increasing pressure to O_2 transfer at tissue level. Kiesow (1974) has shown that the *in vitro* oxygen–haemoglobin dissociation curve flattens to the left with increasing pressure, although the effect is small at current depths. The *in vivo* response may not be so kind. Coupled with this is the inhibiting effect of He on phosphorylase reactions and its interference with cyanide–cytochrome oxidase bonds, so that metabolism becomes less efficient (South & Cook 1953; Harrison & Solomon 1975; Shanklin & Lester 1972; Rinfret & Doebbler 1961; Shreiner *et al.* 1962). In view of this, it is not surprising that lactic acid appears in the blood sooner after the start of exercise in normal subjects exposed to quite minor pressures of Heliox (Hanson *et al.* 1981) or that there appears to be a base offset of blood lactate levels with higher steady state levels. It is not known whether compensatory adaption would occur with time at pressure, but the ventilatory and cardiovascular changes reverse when decompressing, although at a different rate than when compressing. On arrival at the surface, no residual cardiovascular effects are seen.

Haematology

Of all the numerous haematological parameters studied, only the erythrocyte sedimentation rate, platelets (thrombocytes), and the haemoglobin and associated packed cell volume has shown changes consistent enough to be ascribable to the hyperbaric environment, and the main features of these changes will be given, and their significance assessed.

In general, the erythrocyte sedimentation rate has correlated well with type I decompression sickness throughout a whole series of saturation dives between 180 and 660 m s.w. with a variety of breathing gas mixtures, the pattern of change being one of increased sedimentation during the decompression phase with maximum values to be found at or near to the surface. Of 25 subjects, 23 (92%) showed varying degrees of response, with values as high as 50 mm above the pre-dive baseline; 17 responses were considered significant and were associated with 12 incidents (70%) of type I decompression sickness. Six responses were thought not to be significant but still associated with two cases of type I and one of type II decompression sickness. No decompression sickness problems were encountered with the two subjects who showed no erythrocyte sedimentation rate changes.

Sicardi (1970) was the first to report a loss of platelets in human subjects immediately after decompression. This was followed (Martin & Nichols 1972) by observations that the circulating platelet count continued to fall for about 72 h after a 1 h exposure to a simulated depth of 100 ft (30.5 m s.w.) and a 2 h 3 min staged decompression, which, in over 200 open-sea dives, had proven exceptionally free from decompression incidents.

Since then other investigators have variously confirmed the observation or, in some instances, found no significant change, but we have consistently noted a depression of circulating platelets during the decompression phase of the deeper oxygen–helium and Trimix saturation dives. The magnitude of the depression varied from dive to dive and between subjects, but was usually between about 10 and 30%, and was seen in both the incident-free dives as well as those that presented a decompression problem.

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Haemoconcentration, as indicated by an increase in packed cell volume, or by estimation of blood volume, has been a frequent observation associated with decompression sickness in animals. In man these changes have been reported to occur in two directions with both increase and decrease in values being noted. Our own investigations have generally shown a tendency to increase initially while at maximum depth, followed by an insidious reduction, throughout the decompression phase, in both the haemoglobin and packed cell volume. The loss on previous dives, although observed, had not been sufficient to cause concern. However, during the long decompression from 660 m s.w. both subjects lost, on average, 2.5 g from their level before diving and almost 4.0 g from the highest level recorded at maximum depth.

Data collected indicates a pattern of fairly consistent increase in the erythrocyte sedimentation rate of human subjects during decompression from saturation dives. However, the non-specificity of the reaction makes interpretation difficult. The complex mechanism of sedimentation can be influenced by many factors, including the presence of increased protein fractions such as fibrinogen and globulin; it is increased in anaemia and in all cases of cell destruction, which occurs in trauma, toxic conditions and infections, but only on rare occasions has it been possible to demonstrate a measurable increase in protein fractions. Correction for the anaemia seen in the deeper dives makes no significant difference to the erythrocyte sedimentation rate results. There is little or no relation between sedimentation and the degree of aeration, or loss of carbon dioxide from the blood, so it would seem that gaseous exchange is not such an important factor. The partial pressure of oxygen does not seem to contribute to the change, as this has varied from 40 kPa in the saturation dives, to 80 kPa in a series of non-saturation dives where no change occurred. The duration of exposure to a raised oxygen tension likewise does not appear relevant as this was the same for the dive producing the greatest change as that showing the least. The magnitude of change is, apparently, not depth related, but could be more closely associated with the actual profile used for decompression and, therefore, may be a sensitive indicator of a potential problem even in the absence of overt signs and symptoms of decompression sickness.

Experimental evidence suggests a role for the involvement of platelets in decompression sickness. Adherence and aggregation of platelets to bubble surfaces has been demonstrated and microthrombi have been shown to be present in severe cases of decompression sickness in animals. Despite the consistent thrombocytopaenic response found in man during decompression, information concerning the disposition of platelets in clinical cases of decompression sickness is almost non-existent. It may be that platelets are being used in laying down thrombi in the microcirculation, particularly the canaliculi of the long bones, and thus playing a part in long-term effects of repetitive diving, such as dysbaric osteonecrosis.

The loss of haemoglobin has only been considered of significance in the prolonged deeper dives and the reason for the response is not yet understood. It seems unlikely that it is a result of repeated blood sampling, as the amount taken from a subject is rarely more than 20 ml every 48 h, hardly enough to cause an apparent anaemic response in the healthy adult male unless accompanied by other causes such as increased red cell destruction or a transient depression of erythropoiesis due, perhaps, to prolonged exposure to an increased partial pressure of oxygen. It may be the opposite, compensatory, reaction to the polycythaemia of hypoxia and, while the oxygen pressure is elevated, the subject remains asymptomatic, but on return to the surface the reduced oxygen carrying capacity of the blood becomes evident with the consequential feeling of gross fatigue invariably experienced after these dives.

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Decompression from deep saturation

The design and assessment of decompression profiles from great depths (over 180 m s.w.) is hampered by the small number of trials and the lack of an independent method of detecting either an excess of undissolved gas or impending decompression sickness.

Currently, the 'end point' of a profile under trial occurs when a subject reports symptoms of decompression sickness, which, in the opinion of the attending medical officer, are sufficiently severe to warrant therapeutic treatment (by halting the ascent, possibly followed by recompression or raising the proportion of inspired oxygen, or both). Subsequent reduction or relief from pain (again, as reported by the subject) may be taken as confirmation of the diagnosis of decompression sickness.

With such an imprecise and subjective 'end point' the evolution of deep decompression profiles and associated therapeutic procedures has been largely empirical. Nevertheless, while a more objective indicator of an excess gas phase would certainly assist in the correct assessment of a given decompression profile, it would probably not reveal its source, depth of separation from solution and ensuing history, information necessary to enable the unsafe portion of an unsuccessful profile to be identified and amended. This can be stated because both static and circulating free gas has been detected ultrasonically (Powell *et al.* 1982), and it appears that it can exist asymptomatically for many days (Brubakk *et al.* 1982; Davies 1983) during long slow decompression. Also, and unfortunately, not all cases progress to the decompression sickness end point described above.

The same can be said of the haematological changes that have often been observed on decompression (Hallenbeck & Andersen 1982). In any case, such changes take several days to become significant, sometimes only after decompression sickness has actually occurred. So it is not surprising that, in the early days of deep saturation diving, the immediate portion of a profile associated with a case of decompression sickness was amended, rather than the deeper stages.

There have been instances where an unsuccessful model, as proved by a failed profile from one depth, was modified and used to generate a profile, which also failed, for an even greater depth. Such extrapolation from an incomplete data base containing known errors is inherently unsound. An example of this occurred at the Physiological Laboratory in 1969–70 in attempting to develop a mono-exponential profile for a depth of 457 m s.w. (Morrison *et al.* 1976).

A more systematic approach using variable-depth constant-time stages followed some years later (Barnard 1976), but was abandoned when attempts failed to extend the method beyond 100 m s.w. After many more trials, a purely empirical semi-parabolic profile evolved, and was used for depths to 250 m s.w. (Vorosmarti *et al.* 1978). Although this profile is currently used by the Royal Navy, it is certainly not free of minor symptoms of joint pain (niggles), particularly in the final stages of an ascent from 200 to 250 m s.w. and linear extrapolation to 300 m s.w. depth (Hennessy 1980).

If a gas phase is distributed in the tissue responsible for such marginal symptoms of decompression sickness, an elementary first order analysis predicts that for a constant 40 kPa oxygen partial pressure, the ideal profile will be a continuous linear ascent, at a rate of 28 m s.w./d (Hennessy 1980). Workman (1969) also predicted a linear profile by classical decompression theory. Clearly, at present, more detailed mathematical modelling of gas exchange in tissue is unwarranted, and there appears to be little point in performing a curvilinear

as opposed to a linear profile, however derived, theoretically or empirically, as long as there does not exist an independent means of either testing a model, or of refining the coarse and highly subjective end point of decompression sickness. The 28 m s.w./d linear profile was introduced into the Physiological Laboratory deep dive programme, starting with Dive 8 in February 1978. The chief constraint to the ideal continuous ascent was the nightly hold of 8–10 h, which, it was felt, would avoid the possibility of decompression sickness arising in sleeping divers. This meant that to maintain the average rate of 28 m s.w. in 24 h, the instantaneous rate had to be accelerated during the daily running time. Further analysis suggested that a rate not exceeding 4 m s.w./h might be tolerable, although it was uncertain how long such a rate could be maintained (Hennessy 1980). So the 28 m s.w. ascent was spread over 7 h running time each day and appeared to provide adequate safety while offering a rationale by which future decompression experiments could be designed. If the profile should fail it would not of course be possible to identify whether the daily (average) rate or the hourly (instantaneous) rate had been too fast. However, holding the 28 m s.w./d rate (and the oxygen partial pressure) constant, and varying the hourly rate over a series of trials appeared to be a more systematic approach than that of previous designs.

The daily accelerated portions of the ascent were an attractive feature, because over a very long ascent they could be viewed either as amplifying a marginal gas phase or increasing the rate of inert gas elimination (these two extremes are inherent in a standard staged decompression).

Table 2 gives a summary of all those decompressions where the 28 m s.w./d linear profile was used. On Dive 9B both subjects reported joint pain at 38 m s.w., after a trouble-free ascent from 540 m s.w. This suggested that the 4 m s.w./h rate was perhaps too fast and a 1.75 m s.w./h rate was attempted on Dive 10A from the modest depth of 180 m s.w. This resulted in a case of thigh pain at 12 m s.w. on the ascent, but may have originated as deep as 90 m s.w. This was rather surprising because the actual profile was very similar to the well proven U.S. Navy schedule in the major portion of the ascent. On Dive 12B the 28 m s.w./d rate was slowed to 24 m s.w./d, but at 2 m s.w./h, to allow for a constant 10% (by volume) proportion of N_2. After an uneventful 19 d run of 483 m s.w. from 660 m s.w., a remarkable case of neurological decompression sickness (in the form of motor and sensory disturbance of the left arm and leg) occurred at 117 m s.w. in one subject (this is the first recorded case of serious neurological involvement at such a slow rate of ascent). A possibly quite unrelated incident occurred two days before this event: the subject had been complaining of sinus congestion, and decompression had been halted for a few hours while he received the antihistamine drug, Histryl. Although congestion was relieved, and the decompression re-started, two side-effects were reported by the subject (sedation, parasthesia around the mouth and left arm). It is not known whether vaso-constriction or vaso-dilation, another known side-effect, occurred.

On Dive 81A to 540 m s.w., the last deep dive in the present series, a 19 d clear run of 529 m s.w. was followed by another remarkable case of neurological decompression sickness (in the form of visual disturbances), at about 11 m s.w., in the same subject that had a central nervous system event in Dive 12B. Over the preceding few days he had reported chest discomfort, which may have been unrelated to the ensuing central nervous system event.

Unlike the continuous decompressions done on the Duke University ATLANTIS series, the profiles in table 2 have not been plagued by intermittent joint pains in the deeper portions.

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In fact they have been rather successful in permitting subjects to safely attain shallower, and more manageable, depth zones. Nevertheless, two peculiar cases of neurological disturbance occurred on this series and it is possible that this may be due to the cumulative effect of several weeks of circulating microbubbles. As was pointed out by Hennessy (1980), a supersaturated tissue (associated with ordinary decompression sickness) may not be able to 'capture' a

TABLE 2. TRIALS OF THE 28 m s.w./d EXPERIMENTAL PROFILE

date	dive number†	saturation depth–terminal depth	rate m s.w./h	symptoms
February 1978	8	420–30	4	none
March 1978	9A	180–10	4	none
May 1979	87	200–0	4	none
May 1979	9B	540–38	4	both subjects had knee pain or niggles at 38 m s.w.
September 1979	10A	180–0	1.75	1 subject had right mid-thigh niggle at 12 m s.w. and 2 h after surfacing
February 1980	11	300–180‡	4	none
November 1980	12B§	660–177	2	1 subject had a central nervous system event at 177 m s.w.
November 1981	81A	540–11	2	1 subject had a central nervous system event at 11 m s.w.

† Dry chamber series: two subjects per experiment, except Deep Trials Unit Dive number 87, which had three subjects.

‡ A 41 h hold was inserted at 180 m s.w. to do thermal studies.

§ The gas mixture on this dive contained constant 10% (by volume) N_2, and the average ascent rate was slowed to 24 m s.w./d.

circulating microbubble at great ambient pressures, even if the capillary residence time may normally be long enough at a lower pressure. This is simply because the rate of increase, or decrease, of bubble diameter as it passes through tissue is proportional to the reciprocal of the ambient pressure and to the tissue–bubble inert gas gradient, both of which are very small quantities on the slow ascents from deep saturation exposures. However, if the capillary closes during transit, the microbubble will indeed by captured, but will grow exceedingly slowly, leading to a virtually stabilized gas distribution in surrounding tissue.

Before abandoning the 28 m s.w./d rate, perhaps a return should be made to the 4 m s.w./h rate to reveal the existence of such a gas phase at an earlier stage of the ascent.

DISCUSSION OF FINDINGS

Simulated diving comprises a compression phase, a time at pressure, and a decompression phase. The breathing gas compositions, and thermal environments, are superimposed on this pressure–time course. These physicochemical features of the hyperbaric experimentation will be used to order the principal physiological findings and assess their significance. It is appreciated that events occurring, for example, during the time at pressure, clearly influence events occurring during the subsequent decompression, and also changes in one physiological system must interact with other physiological systems, but despite this complex interplay of environmental and physiological factors there are some helpful general observations to be made by using the above conceptual framework.

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Thermal exchange

Many neurophysiological mechanisms were seen to be affected by the grossly altered hyperbaric helium–oxygen environment and it was feared that the subject's estimation of his thermal status might be quite misleading, thus contributing to an overall loss or gain in total body heat. However, as seen earlier in this paper, there is no doubt now that throughout the whole dive the volunteer's estimate of a comfortable temperature is soundly based. Control of total body heat and the inner temperatures of the body are accurately maintained.

It is quite possible to account for the serious body weight changes that have often been reported elsewhere, but although it is now established that no direct problems need to be encountered in the maintenance of energy and fluid balances at high pressures, to achieve this desirable result there have to be appropriate compensatory shifts in other physiological systems such as peripheral blood flow. From a practical standpoint it is reassuring that the central control mechanisms are able to organize all these compensatory moves, thus indicating they are relatively unimpaired to pressures of the order of 6 MPa.

Compression

As has been pointed out (Török 1982), the significant neurological changes that can be encountered when compressing on helium–oxygen at depths in excess of 160 m s.w. may be grouped under four systems; motor, vestibular, cerebral cortex, autonomic; each with essentially independent courses of development.

Of all the signs and symptoms it is those generated by disturbances in the vestibular system that are most distressing to the volunteers. Avoidance of these problems seems to transform their whole approach to the work programme. Fortunately it is the vestibular sensory system that seems to be totally dependent upon the compression profile, and this additional observation further confirms Török's groupings based largely upon case histories. This vestibular form of compression sickness bears the same kinds of relations to compression as decompression sickness does to decompression (see table 3).

The presence of 10% (by volume) nitrogen in the helium–oxygen breathing mixture also effectively suppresses the vestibular problems, despite the use of rapid compression time courses, for example, 420 m s.w. in 7 h, but hastening the compression in this manner can provoke serious problems of narcosis that are far worse than would be expected from the partial pressures of nitrogen present in the breathing gas.

Tremor of intentional movement is not easy to suppress, but there is little doubt that the subjects adapt to performing fine manipulative tasks, for example, venepunctures. The observation that the tremors worsen after a good night's sleep shows how routine physiological events can affect the findings.

It seems that compression provokes signs and symptoms that are truly sensitive only to changes of pressure, but has some manifestations that are only partially influenced by the pressure–time courses used. It may be that these resistant forms of compression sickness are more accurately designated as pressure sickness. There is a remarkable synchronization between the appearances of many of these various neurological signs and symptoms. On compression rapidly to 150 m s.w. depth, it is almost unknown to meet any abnormalities, whereas beyond 200 m s.w. everyone is involved to some extent. This strongly suggests a common aetiology for many of these diverse responses.

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Table 3. Comparison of factors affecting compression sickness and decompression sickness

compression sickness	*decompression sickness*
provoked by positive changes in pressure	provoked by negative changes in pressure
reversed by decompression	reversed by recompression
can exhibit a several-hour delay period before symptoms become apparent	can exhibit a several-hour delay period before symptoms become apparent
sensitive to gas composition	sensitive to gas composition
can be provoked by gas switching only	can be provoked by gas switching only
signs and symptoms mainly from vestibular sensory system, but from somnolence (microsleep) also.	several separate body systems can be involved

At pressure

Once at a steady pressure the changes caused by the compression generally follow a gradual, not necessarily smooth, path of being less noticeable with passage of time. On some occasions it is possible to arrive at maximum depth with only minimal signs of compression sickness, but after the passage of two or three hours ill effects can overtake the subjects. On the few occasions when this has happened it has always been abundantly clear from the next experiment that the compression to that depth had been at fault. It is worth remarking that the success of short (2 or 3 h) 'excursions', for example, from 365 to 488 m s.w. (Lambertsen 1976) could be attributed to this delayed onset of compression sickness.

Even with a pressure–time course that minimizes compression sickness at pressures in excess of 300 m s.w. when breathing helium–oxygen, there are decrements in cognitive and psychomotor performance (Logie & Baddeley 1983). However, individual variations are very large and some activities, for example, associative memory, can remain hardly affected, whereas mental arithmetic ability can suffer large decrements. The integrated picture is difficult to assess from these various tests unless, as happened following rapid compression on Trimix to 420 m s.w., both subjects were unable to cooperate sensibly on any of the tests! The indications are that despite best endeavours with the compression phase there is a worsening of performance with increase in pressure.

The changes in heart rate and cardiac output, while breathing Trimix or helium–oxygen gases, are beginning to show a serious trend, but even more potentially troublesome are the biochemical findings at pressures in excess of 300 m s.w. The latter are also independent of the nature of the breathing gases used, but a very high partial pressure of helium is always present in all these experimental deep dives.

It is not possible to distinguish between the effects of hydrostatic pressure and those of dissolved gas. Clearly the latter can be remarkably influential, as shown by the presence of comparatively small concentrations of nitrogen gas in mixtures of helium–oxygen.

Decompression

As outlined earlier, the decompression procedures followed a pattern of slow linear decompression during daylight hours and an overnight halt, for sleep purposes, at constant pressures. The remarkable and morale-sustaining success of this procedure during the high

pressure period of the decompression contrasts with the aggravating frequency of decompression sickness during the low pressure levels of the procedures.

Some large and easily measurable haematological changes were observed during the decompression. These were true decompression phenomena in the sense that they only appeared during the decompression phase, and returned towards normal whenever decompression halted. It is considered that some of these changes could be used as simple and objective measures of the effectiveness of a decompression profile, but further work will be necessary to establish proper levels of confidence.

All the changed responses seen in the course of this experimentation reverted to normal, either during the decompression or afterwards. The practical conclusion of this work is, therefore, that diving to depths of 540 m s.w. by using a helium–oxygen breathing mixture can be effective and safe.

Continuous and prolonged human experimentation in potentially dangerous circumstances demands participants with high levels of competence, persistence and dedication. We thank all those who helped in these deep diving trials. Particular mention is necessary for the volunteers, especially M. S. English, P. J. Atherton and R. S. McKenzie; the helpful guidance from the Ethical Committee, under the Chairmanship of Professor W. R. Keatinge; the drive and dynamicism of Dr R. F. Carlyle and his assistant S. A. Collis; and the physicians 'on call' from the Institute of Naval Medicine. Above all we thank the high pressure team under the control of E. J. Towse for acting as a stable, central hub, around which all our activities revolved.

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Discussion

W. D. M. Paton, F.R.S. (*Department of Pharmacology, Oxford, U.K.*). I would like to comment on the system of classification of the h.p.n.s. used in the presentation. 'Autonomic' is a word usually applied to the sympathetic and parasympathetic systems, yet responses involving voluntary muscle were included. Somnolence is included under 'cortical', although I do not think it is known to originate in the cortex. Symptoms included under 'vestibular' might also originate elsewhere, for instance, the area postrema. The danger of classifications is that they tend to condition subsequent thinking as regards the mechanisms involved. Might not a more non-committal classification be safer?

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Z. Török. The proposed classification of the most important symptoms and signs in man into motor system, vestibular sensory system, autonomic and cerebrocortical groups assigns systems likely to be their cause. For somnolence, the classification was cortical on the basis of its accompanying e.e.g. with predominant slow waves. The underlying chain of events may indeed start elsewhere, i.e. in the brain stem reticular formation. Changes in breathing pattern, such as hyperventilation, were grouped as autonomic, as they are likely to reflect the subject's state of arousal. Lightheadedness may also have arousal as the most likely cause; admittedly, other explanations are possible.

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