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Probing the Limits of Human Deep Diving [and Discussion]

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Probing the limits of human deep diving

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Divers breathing compressed air are restricted to 45 m depth because of the narcotic effects of nitrogen and toxic action of oxygen at increased pressures. Substitution of oxygen–helium for compressed air has permitted divers to reach 600 m. However, at depths greater than 160 m, signs and symptoms of the high pressure nervous syndrome (h.p.n.s.) occur, with tremors, myoclonic jerking, nausea, vomiting, fatigue, somnolence, e.e.g. changes, dyspnoea, and poor sleep with nightmares. It has been the objective of this Laboratory to ameliorate the symptoms of pressure-induced h.p.n.s. by the addition of small amounts of 'narcotic' nitrogen to the oxygen–helium mixture to form the Trimix breathing gas. In 1973, comparative experiments with oxygen–helium and the same divers, during compressions in only 33 min to 219.5 m and 305 m, showed such Trimix to be effective with 10% (by volume) nitrogen. Simulated dives, termed ATLANTIS, have been made with Trimix over the last 4 years to depths in excess of 610 m for 11 days, 650 m for 4 days and 686 m for 1 day. The objectives were to determine the effects of either slow or rapid rates of compression, and either 5% or 10% (by volume) nitrogen in Heliox, on the presence of h.p.n.s. or nitrogen narcosis. Measurements were made of intellectual and psychomotor performance, electrophysiological function of the brain and reflexes, lung and cardiovascular function, including arterial gas analysis at rest and work, blood chemistry and psychiatric and psychological status. The results permit the conclusion that divers may be compressed safely to depths as great as 686 m. The technique requires a slow exponential compression over days, with frequent stages lasting 14 h or more, the use of 5–8% (by volume) nitrogen in Heliox and careful selection of the divers.

Introduction

The wide international recognition given to probing the limits of outer space in the last decades by the United States and Russia has overshadowed an equally challenging but an even more formidable physiological and medical barrier: probing the limits of human exposures deep under the ocean, which forms 70% of this planet. There is no practical working environment with a more severe and complex composite of physiological stresses than that encountered by the modern deep diver. The human deep diver, unlike the astronaut who is well protected by elegant engineering from the environment, suffers from an increasing number of physiological stresses as the hydrostatic and gas pressures increase (Bennett & Elliott 1982). He also becomes 'physiologically trapped' by this environment because it still requires 7–10 days to decompress a diver to sea level from a deep-saturation oxygen–helium dive of say only 305 m (1000 ft), and 32 days from 686 m (2250 ft), a time that is longer than it takes to return an astronaut from the Moon.

Compressed air limits

In the early decades of this century, Sir Leonard Hill and his colleagues (1906, 1932, 1933) probed the limits of diving with compressed air. At 91 m (300 ft) they noted compressed-air-induced signs and symptoms of narcosis, with slowing of the process of cerebration, difficulty in assimilating facts and making rapid decisions, including 'semi-loss of consciousness', and amnaesia on return to the surface; the severity of which, for all these, increased with depth. This restricted diving to about 90 m (300 ft), albeit with the diver dangerously intoxicated. Most navies restricted diving with air to some 45 m (Bennett 1982*a*). Even the oxygen in air was a major difficulty. The increasing partial pressure of oxygen, as noted by Bert (1878) as early as 1878, subjects the body to the risks of sometimes fatal convulsions or pathological changes in the lung (Smith 1899) and must be kept no higher than 50 kPa (Clark 1982).

Yet it was not until between 1935 and 1939 that Behnke *et al.* (1935) inferred that compressed air intoxication was due to the increased partial pressure of nitrogen, which acted as an inert gas narcotic, or that substitution of helium for the nitrogen would alleviate the problem.

This was based on the Meyer–Overton theory applied to general anaesthetics that there is a parallel between the affinity of an aliphatic anaesthetic for lipid and its narcotic potency. Thus, compared with nitrogen, the use of the 4.26 times less lipid soluble helium, implied that depths of as much as 305 m (1000 ft) should be possible without incapacitating narcosis. This also would depend on keeping the oxygen partial pressure low (50 kPa) so as to prevent oxygen toxicity. However, this hypothesis was not to be tested until nearly 30 years later.

In 1962 a young Swiss mathematician, Hannes Keller, then confounded researchers in diving medicine, by reaching a record 305 m (1000 ft) while breathing oxygen–helium with a remarkably fast compression rate of 100–150 kPa/min and, after only a rather brief 5 min at depth, an even more remarkable decompression to the surface in only 270 min (Keller & Bühlmann 1965). This advance ran parallel with the development of saturation diving technology in the U.S.A. by Captain George Bond (Bond 1966; Workman *et al.* 1962), in which the diver lives for days at pressure, saturating his body with the gases he is breathing and requiring only a single decompression to the surface. Over the last twenty years, helium and saturation diving methods have been used extensively for both commercial and military purposes to probe the physiological limits of human deep diving (Bennett 1982). In so doing a new formidable barrier to further progress was recognized in 1964, called the high pressure neurological syndrome (h.p.n.s.).

The high pressure nervous syndrome (h.p.n.s.)

The h.p.n.s. was first noted by the author in man during a series of oxygen–helium deep dives in 1964 and 1965 at the then R.N. Physiological Laboratory (R.N.P.L.), Alverstoke. Navy volunteers were compressed to 183 m (600 ft) for 4 h and to 244 m (800 ft) for 1 h at compression rates of 30.5 m/min (100 ft/min). Surprisingly, the divers showed decreases of 18% in arithmetic ability and 25% in the ball-bearing test of fine manual dexterity at 183 m, which at 244 m were much worse, at 42% and 53% respectively (Bennett 1965, 1967; Bennett & Dossett 1967), and were accompanied by marked tremors of the hands, arms and torso, dizziness, nausea and vomiting. Most of the divers recovered in 90 min.

Nevertheless, their initial state was such that projections to 305 m (1000 ft) under similar conditions implied that the divers might be severely incapacitated. Bennett termed the condition 'helium tremors'. Brauer *et al.* (1966) and Miller *et al.* (1967), however, working independently with animals, in the U.S.A. and U.K., on the inert gas narcosis problem and pressure antagonism of narcosis, noted that increased hydrostatic pressure itself may pose a significant problem and termed the tremors and convulsions seen in rats and other animals at increased pressures the 'high pressure neurological syndrome'. This has now come to be called the 'high pressure nervous syndrome' or h.p.n.s.

H.p.n.s. in man is characterized by dizziness, nausea, vomiting, postural and intention tremors, fatigue and somnolence, myoclonic jerking, stomach cramps, increased slow wave and decreased fast wave activity of the e.e.g., decrements in intellectual and psychomotor performance and poor sleep with nightmares. The severity is influenced by both the compression rate and the degree of increased pressure. So the syndrome is enhanced by rapid compression and decreased by an exponentially slow rate of compression, with stages at interim depths to permit adaptation. Some divers appear to be more susceptible than others (Hunter & Bennett 1974; Bennett 1982).

H.p.n.s. in oxygen–helium diving

It was not long before the possible limitations of h.p.n.s. at 305 m (1000 ft) were to be challenged. In 1968 the U.S. Navy made the first saturation dive to 305 m for 77 h 22 min with a very conservative total compression time of 24 h at the new diving research facilities of the F. G. Hall Laboratory at Duke Medical Center, U.S.A. Virtually no h.p.n.s. was seen with this very slow compression rate (Summit *et al.* 1971). At the same time a French commercial diving company, COMEX, conducted a simulated oxygen–helium dive with two men compressed in 2 h to 363 m (1190 ft). However, it was aborted after only 4 min at depth, owing to dizziness, tremors, nausea and the first observations of large increases in e.e.g. θ-activity (4–6 Hz). This was accompanied by depression of α-activity (8–13 Hz), intermittent bouts of fatigue and somnolence and sleep stages 1 and 2 termed 'microsleep' (Brauer 1968). The severe nature of these signs and symptoms led Brauer to postulate that these might be due to a physiological barrier to deep diving. Yet only a few months later, three Swiss divers, with the principles used earlier by Keller, were compressed in a joint Swiss and British study to 305 m (1000 ft) in 1 h with only brief and minor h.p.n.s. on arrival. They remained there for 3 days and made 2 h excursions in water, to 350 m (1150 ft) on the second and third days (Bühlmann *et al.* 1970) without difficulty.

On the basis of the excellent condition of these divers at 305 m and even 350 m, Bennett challenged the existence of a barrier and conducted, in 1970 at the R.N.P.L., the first extensive physiological investigation of h.p.n.s. during compression in stages over 3 days to 457 m (1500 ft) for 10 h (Bennett & Towse 1971*a*, *b*; Bennett & Gray 1971; Bennett *et al.* 1971; Morrison & Florio 1971). Compression (figure 1) was at 5 m/min with 24 h stages at 183 m (600 ft), 305 m (1000 ft) and 396 m (1300 ft), and 1 h stages at 335 m (1100 ft), 366 m (1200 ft) and 427 m (1400 ft). Mild symptoms of h.p.n.s. such as tremors, some nausea and myoclonic jerks were noted during the compression to each stage, but tended to dissipate at stable depths. For the first time on-line frequency analysis showed that θ-activity markedly increased during each compression, but returned to more normal levels at the stages (figure 2). Owing to the hand tremors, psychomotor tests indicated decreased efficiency, but arithmetic tests of

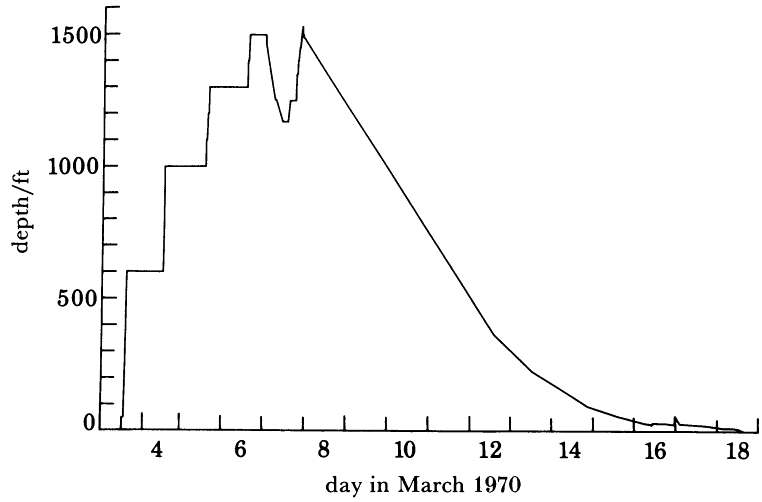

FIGURE 1. Compression–decompression profile for two men compressed to 457 m (1500 ft) for 10 h at R.N.P.L. Long and short stages during compression are shown. During decompression vestibular decompression sickness occurred in one diver requiring recompression treatment. A further mild knee bend required recompression at 9 m (30 ft). (After Bennett *et al.* 1971 *a*.)

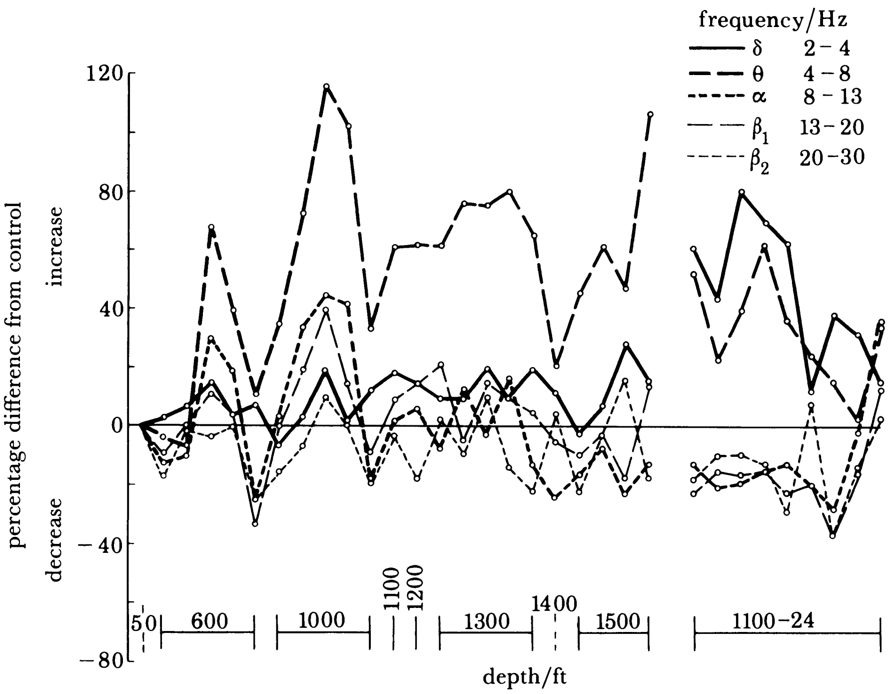

FIGURE 2. Percentage change in frequency analysis of the e.e.g. (eyes open) of one of the two divers during the 457 m (1500 ft) oxygen–helium dive at the R.N.P.L. Compression at each stage initiated a marked rise in θ-activity (4–8 Hz), which continued to increase for 6 h and then, over 12 h, returned to lower values but still above the normal level. (After Bennett *et al.* 1971 *a*.)

cognitive function were unimpaired (figure 3). The two divers varied significantly, however, in their sensitivity.

It was now clear that h.p.n.s. is a function of both compression rate and hydrostatic pressure. Further that with slow compression and stages for adaptation it is possible for suitable people to dive to considerable depths with only minimal h.p.n.s.

Later in 1970 the COMEX company extended these methods with two men in a dive called Physalie V. This used an exponential compression profile with stages of 3 d 83 min to reach 520 m (1706 ft) for 77 min, and was followed in 1972 by Physalie VI with compression in 7 d 8 h to 610 m (2001 ft) for 80 min (Fructus 1972). A longer time of 50 h was spent at 610 m in the final oxygen–helium COMEX 1974 dive (Rostain *et al.* 1976; Fructus *et al.* 1976).

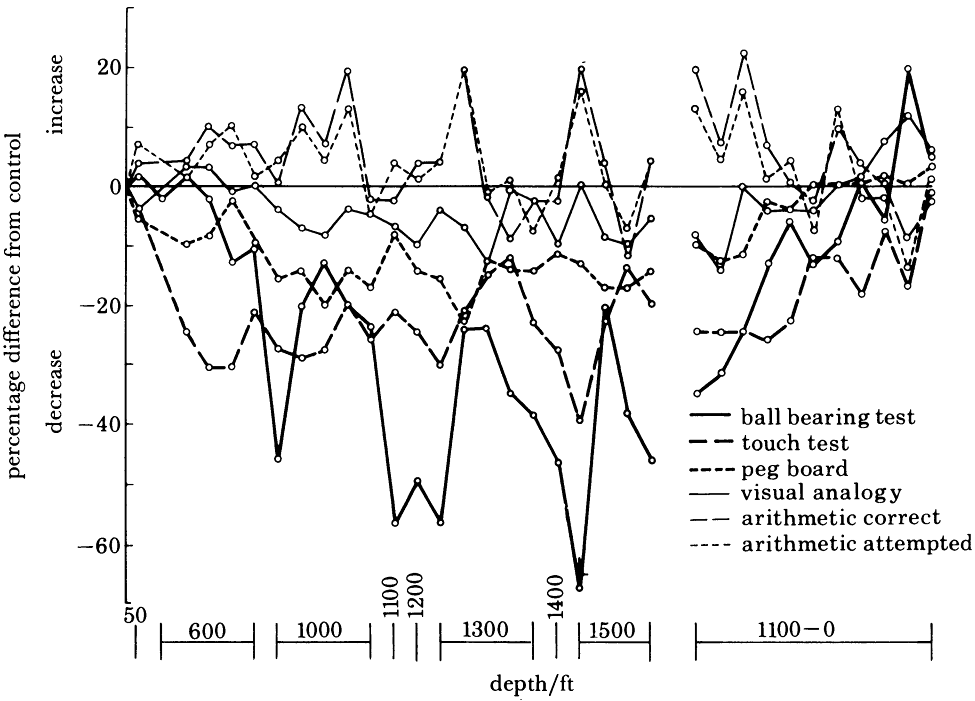

FIGURE 3. Percentage change in intellectual and psychomotor performance tests during 457 m (1500 ft) oxygen–helium exposure for one of the two divers. Intellectual tests were unaffected but psychomotor efficiency was impaired. The decrement in the ball-bearing test correlates with the onset of tremors elicited by compression. (After Bennett *et al.* 1971 *b*.)

In the latter dive, two men were compressed in 10 d 21 h and for the first time stayed for 50 h at this depth, much more than the previous brief minutes. Tremors were present and increased by 160–190%. E.e.g. changes, with dramatic 2000–4000% increases in θ-activity were seen from the anterior brain and 500–1000% in the middle regions. Fatigue and 'microsleep' also occurred, with suppression of sleep stages 3 and 4, vivid dreams and out-of-body experiences.

It was now evident that at depths in excess of 457 m (1500 ft), h.p.n.s. was providing definite limitations to functional efficiency and the health and safety of the diver. This was confirmed by two further dives in 1979, one in England at the R.N.P.L. (now renamed A.M.T.E./P.L.) and the other at the U.S. Navy Experimental Diving Unit.

The British dive (A.M.T.E./P.L. 9) involved compression of two divers to 540 m (1771 ft) in 3 d 5 h (Hempleman & Harris 1979, personal communication; Bennett 1982) with 2.5 d at maximum depth. After 420 m (1377 ft) there was marked nausea, tremors, dizziness, vomiting and anorexia. The U.S. Navy dive involved compression of six divers to 549 m (1800 ft) over 3.75 d from an 8 d saturation stage at 198 m (650 ft). H.p.n.s. symptoms were severe and varied with individual susceptibility. They included fatigue, nausea, vomiting, aversion to food with 8% weight loss, stomach cramps, diarrhoea, myoclonic jerking, dyspnoea and nightmares that were often associated with flying (Spaur 1980).

Clearly the strategy of slow exponential compression with stages, adaptation with time at depth, selection of divers or excursions from shallower depths was not providing the complete solution to the limits on deep diving imposed by h.p.n.s. (Bennett 1975, 1980).

Control of h.p.n.s. by Trimix (helium–nitrogen–oxygen)

Johnson & Flagler (1950) noted that the anaesthetic effect of ethyl alcohol on tadpoles could be reversed by application of 150 atm hydrostatic pressure. Lever *et al.* (1971) made similar observations in mice; the loss of righting response at increased pressures of nitrogen could be restored by increased pressures of helium. Bennett *et al.* (1967), measuring surface tension changes on a lipid monolayer exposed to increased pressures of inert gases, found that nitrogen, argon, carbon dioxide and oxygen caused a fall in surface tension, but that helium and neon indicated the opposite, an increased tension. These findings prompted Bennett *et al.* (1974) to initiate a series of human studies using the reverse phenomenon (that is, addition of nitrogen to the oxygen–helium) to antagonize the effects of pressure and prevent the h.p.n.s. Based on the surface tension studies, divers were compressed rapidly to 219 m (720 ft) in 20 min with 25 % (by volume) nitrogen, 50 kPa oxygen and remainder helium (a Trimix). The same men also were exposed at a later time to 305 m (1000 ft) in a rapid 33 min compression with a Trimix of 18% (by volume) nitrogen (560 kPa N_2 in both studies).

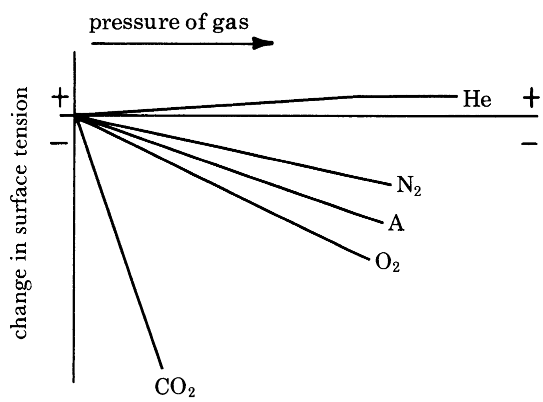

Figure 4. Changes in surface tension in an egg phospholipid monolayer exposed to increased pressures of gases. N_2, O_2, air and N_2 all show a fall in surface tension, which indicates an expanding monolayer. Helium shows a rise in surface tension, which indicates a constriction of the monolayer.

Control studies also were made by the same divers in oxygen–helium at these two same depths, and also with compressed air at 61 m (200 ft). The extensive battery of psychological and neurophysiological tests indicated much improved ability in the presence of Trimix, which suppressed the tremors, dizziness and nausea, but at the cost of euphoria and other indicators of nitrogen narcosis.

Accordingly, on the basis of the Gibbs adsorption equation, a mathematical model was developed (Simon *et al.* 1975) to compute the correct percentage of nitrogen to be added to oxygen–helium to prevent, on the one side, h.p.n.s. (increased monolayer surface tension) or, on the other, nitrogen narcosis (decreased monolayer surface tension). The model predicted

10% (by volume) for nitrogen and for nitrous oxide 0.5% (by volume) or hydrogen 16% (by volume).

The efficacy of Trimix 10 was proved in 1974 with 5 divers compressed to 305 m (1000 ft) in 33 min, which resulted in no performance decrement, no narcosis, tremors or other signs or symptoms of h.p.n.s. (Bennett *et al.* 1975). Underwater work was done for 44 min in 13.3 °C

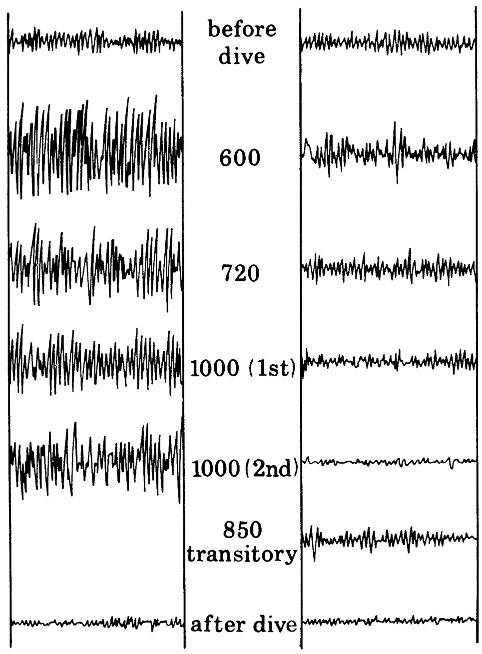

FIGURE 5. Postural tremor from an accelerometer on the index finger of one of three divers compressed to 305 m (1000 ft) in 27 min with 6 min of stages. Results from the helium–oxygen dive are on the left; those from the Trimix dive (18% (by volume) N_2) are on the right. He–O_2 induced marked postural tremor. At 183 m (600 ft) in the Trimix dive, nitrogen was introduced and the tremors were suppressed. At 259 m (850 ft) the diver returned to He–O_2 and the tremors returned. (After Bennett *et al.* 1974.)

water by a diver in a heated suit who noted mild euphoria. Later French work comparing 4.5% or 9% (by volume) nitrogen with slower, 4 h, compressions to 305 m, noted that although there was no significant difference in the performance tests, the lower nitrogen percentage ameliorated the h.p.n.s. with the least euphoria (Charpy *et al.* 1976; Rostain 1976).

THE ATLANTIS TRIMIX PROJECT

These studies led in 1978 to the development of plans at the F. G. Hall Laboratory to study the efficacy of Trimix in the prevention of h.p.n.s. at depths greater than 305 m (1000 ft), where the h.p.n.s. is much more severe. The project, termed ATLANTIS, proposed a series of Trimix dives, first to establish the relation between a specific nitrogen percentage and the rate of compression required to control h.p.n.s., and second to determine the single or combined effects of inspired gas density, hydrostatic pressure and narcosis on respiratory and circulatory parameters, which may be connected with the frequently reported dyspnoea or breathlessness noted by deep divers when working (Lanphier & Camporesi 1982). For each dive one of two nitrogen percentages were selected, either 5% or 10% (by volume) and one of two exponential rates of compression with stages initially to 460 m. One rate was three times faster than any previous dive, for a total of 12 h 20 min, and the other rate twice as slow at 24 h 40 min. Whenever possible, attempts were made to use the same subjects. In the final event four dives were made over 1979–82 as indicated in table 1 with seven divers (table 2), three of whom made more than one dive (Bennett *et al.* 1981; Bennett *et al.* 1982).

Table 1. Project atlantis

dive	N_2 (% by volume)	depth/m	compression time	bottom time/d
ATLANTIS I	5	0–460	12.3 h (fast)	4
ATLANTIS II	10	0–460	12.3 h (fast)	5–6
	10–7.8	460–650	2.3 d	1
ATLANTIS III	10	0–650	4.6 d (slow)	4
	10	650–686	12 h	1
ATLANTIS IV	5	0–650	5.6 d (slow)	2

Table 2. Atlantis divers

profession	age/years	height/cm	mass/kg	ATLANTIS
Duke surgical resident	25	183	71	I
commercial diver	27	185	87	II, III, IV
Duke physician's assistant	40	175	79	I, II
commercial diver	30	173	77	I, III
ex-U.S. Navy U.D.T. student	24	183	74	III
Navy physician and Duke trainee	36	178	77	IV
Duke anaesthesia resident	25	173	64	IV

Extensive scientific investigation was made during these four major deep research dives. This involved sensitive tests of intellectual and psychomotor performance, pulmonary function at rest and work with arterial blood gas data, neurophysiological data involving on-line e.e.g. with frequency analysis, reflexes, haematology and Doppler studies during decompression. Thorough psychological and psychiatric testing of the divers was made before, during and after the dive, and at approximately 6–12 months afterwards, to look for any objective latent effects of these exposures.

In ATLANTIS I in 1979, with 5% (by volume) N_2 and fast compression, h.p.n.s. was experienced during compression, with tremors, nausea, fatigue and, in one diver, vomiting. However, by the day following compression the divers appeared to be functionally normal but a little slower. All experiments were performed, arterial catheters inserted, etc. Some dyspnoea or breathlessness was reported during medium to high bicycle ergometer exercise loads, but the arterial blood gases were within normal limits (Salzano *et al.* 1981). Further, the dyspnoea was worse with oxygen–helium than Trimix.

In 1980, ATLANTIS II, with the same rapid compression but the nitrogen increased to 10% (by volume), was very successful at controlling the debilitating effects of h.p.n.s. seen in ATLANTIS I, such as tremors, nausea, vomiting and microsleep, poor sleep and nightmares. It was possible for divers to perform work at useful levels at 460 m and up to 720 kgf. m min^{-1}

at 650 m. Dyspnoea was largely ameliorated with the 10% (by volume) nitrogen, but at the cost of less efficient gas exchange and some rise in arterial carbon dioxide (Salzano *et al.* 1981). Hyperreflexia, noted in previous Heliox dives, was absent in these Trimix dives (Harris & Bennett 1982).

Indeed, the data at 460 m indicated the divers were in such excellent condition that on the 5th day they were compressed to 650 m in 2.5 d with helium so that the 10% (by volume) nitrogen fell to 7.8%. At this new depth the divers continued to function well for 24 h, although there was a feeling of inspiratory dyspnoea in one subject with obligate mouth breathing. Sleep was good, with less nightmares than ATLANTIS I.

In the arithmetic test requiring simple addition (figure 6), decrements of 50–60% were seen during compression to 400 m and on arrival at 460 m. After 4 d at 460 m these scores had improved to only a 20% decrement. Tests of fine motor performance, such as the ball-bearing test, which required the divers to pick up ball bearings with tweezers and place them in a tube, and gross motor coordination, needed in the handtool test, which requires the use of tools to move nuts and bolts from one metal plate to another, showed much the same result although the decrements were less (figures 7 and 8).

ATLANTIS III was planned to compare the effects of a rate of compression twice as slow as the previous two dives (Bennett *et al.* 1982). The 10% (by volume) nitrogen was equally as successful in controlling adverse effects of h.p.n.s., and on the seventh day the three divers were less tired, more functional and in better condition. There was no dyspnoea, appetite was good, but the shortage of good sleep continued to be a problem. There was evidence of some euphoria, especially in one of the new divers during compression. After 4 d of intensive scientific study at 650 m, the divers were compressed to 686 m (2250 ft) for a day of further research. So the three men spent over 11 d at depths deeper than 600 m.

The performance test data confirmed the value of a slow compression. The large decrements related to compression were not seen (figures 6–7). Instead there was an average decrement of 20% regardless of the depth. However, the addition test (figure 6) showed a marked further decrement at 600 m, 650 m and 686 m. This test, which is very sensitive to nitrogen narcosis, coupled with larger handwriting than normal and the presence of some euphoria, suggested that the 10% (by volume) nitrogen may be too high for such a slow compression. This would agree with the early French dives using 4.6% and 9% (by volume) nitrogen at 305 m and 4 h compression, compared to the 33 min compression to 305 m for the Hall Laboratory dives with 10% (by volume) nitrogen, where the slower compression favoured lower nitrogen.

So ATLANTIS IV, already planned to study the effects of 5% (by volume) nitrogen and slow compression, became more important. This dive took place in 1982 and used virtually the same 7 d compression profile as ATLANTIS III, with one diver the same and two new divers. No feelings of narcosis were present, nor was the increasing decrement in performance seen at 600–686 m in ATLANTIS III (figure 6). Mild temors were seen at this lower nitrogen percentage, however, and this was reflected in the ball-bearing test (figure 7), which at 650 m shows a dramatic decrement of over 60%, nearly three times worse than ATLANTIS III with 10% (by volume) nitrogen. Nevertheless, the diver who had been exposed to ATLANTIS II, III and IV found this the most comfortable compression of them all.

While two of the divers were in remarkably fine condition, the third, a young resident anaesthesiologist, experienced unusual difficulties. At 570 m (1870 ft) he reported being lightheaded and detached with pronouncement of noises and colours, problems in sleeping and

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hyperalertness. He was helped to sleep pharmacologically and improved considerably. However, at the end of the second day at 650 m he had deteriorated and decompression was commenced. As decompression began at the very slow pace of 6 m/h (2 ft/h) he was very happy and uninhibited, with a hyperenergetic state accompanied by incessant talking, visual and auditory hallucinations and delusions of grandeur commensurate with a diagnosis of hypomania.

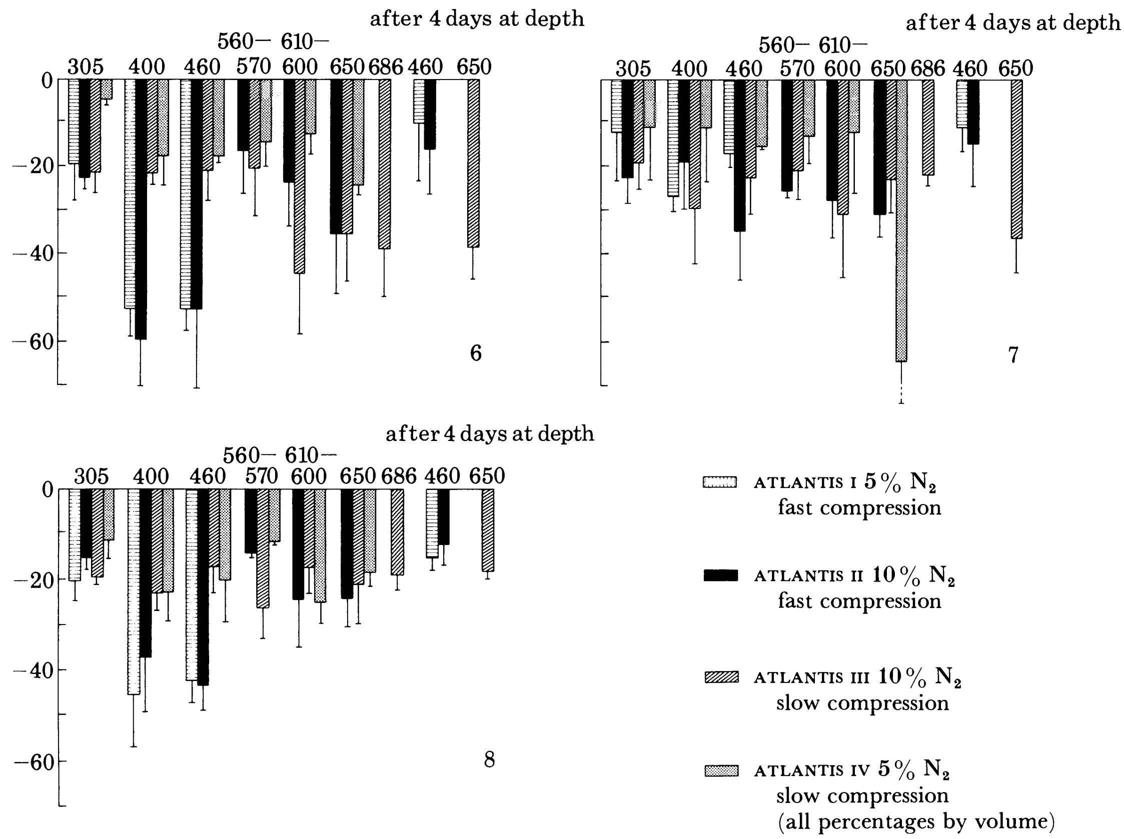

FIGURE 6. Comparison of the mean percentage decrement of three divers for each of the dives ATLANTIS I, II, III and IV at the addition test requiring simple arithmetic. The large decrements at 400 and 600 m for I and II owing to fast compression are evident, as is the increasing decrement for dives deeper than 500 m during II and III and the considerable improvement in ATLANTIS IV.

FIGURE 7. Comparison of the mean percentage decrement of three divers for each of the dives ATLANTIS, I, II, III, IV at the ball-bearing test of fine motor dexterity. There is a tendency for the tests done in less nitrogen to show less decrement, except for ATLANTIS IV when, owing to the presence of visible tremors at 650 m, the test indicates decrements of 60% or more.

FIGURE 8. Comparison of the mean percentage decrement of three divers for each of the dives ATLANTIS I, II, III, IV at the screw plate or hand tool test of motor skills. Large decrements at 400 and 460 m due to the fast compression for I and II are not seen with the slow compressions of III and IV. Otherwise the decrements are around 20% regardless of depth, rate of compression or nitrogen percentage.

This was controlled by sedatives (Valium) at first and at shallower depths more specific drugs such as thorazine and lithium. During decompression from 305 m (1000 ft) the condition resolved and he has now recovered and has returned to his studies.

The psychosis is unusual and not typical of h.p.n.s. It may represent a unique reaction to the specific stresses of confinement inside a 2 m diameter pressure chamber for days, coupled with h.p.n.s., or an aberrant effect of high pressure. However, it does emphasize the need for careful selection of deep divers. In spite of extensive investigations by psychiatrists, clinical psychologists, neurologists and physicians with the most advanced techniques, no indications were obtained of the potential for a psychotic reaction.

In spite of this situation, it can still be concluded that it is possible for selected men to dive in a fit and working condition to 686 m (2250 ft) and probably beyond. This should be done with a slow exponential compression rate with long stages for adaptation. The use of Trimix with 5–8 % (by volume) nitrogen in oxygen–helium is merited to assist control of tremors and other unpleasant effects of h.p.n.s. Considerable further attention is required on sleep, breathing apparatus and lung function, decompression and any possible residual effects of such exposures. To date, over 160 individuals have been exposed to experimental dives over 305 m (1000 ft) and a recent commercial contract had many divers working for 30 d at a time for 6 months at 305 m (1000 ft). These dives resulted in no residual effects consistent with the possible pathophysiology of diving, decompression sickness, h.p.n.s. or hypoxia. However, a cautious approach is advisable and should include regular comprehensive neuropsychiatric evaluations of the divers.

Eventually man certainly will reach a depth beyond which he cannot descend due to physiological limitations. Animals have been exposed to depths as great as 2438 m (8000 ft) and safely returned to the surface. Whether man will ever reach this depth as a diver remains the subject of future research.

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Discussion

Z. Török (*A.M.T.E. Physiological Laboratory*, *Gosport*, *U.K.*). What are the disadvantages of compressing man on Trimix containing 10% (by volume) N_2?

P. B. Bennett. From our ATLANTIS deep dive studies we have learned that use of 10% (by volume) nitrogen in Trimix controls h.p.n.s. but with the risk of undue narcosis in susceptible subjects. The use of about 5% (by volume) nitrogen still permits control of most h.p.n.s. symptoms but without the risk of narcosis. Some postural tremor may be present, however. It seems that with fast compression a higher nitrogen percentage may be required to combat the h.p.n.s.

Z. Török. Therefore one may postulate both, namely too fast and too slow compression, to explain the subjects' poor performance and cerebration seen in the Alverstoke Dive 12b at 660 m. In that dive, typical nitrogen narcosis was seen at 420 m.

P. B. Bennett. I do not think that the problem is caused by too slow compression. The divers in the Alverstoke 12b dive at 660 m were incapacitated because the compression from 420 m was 2.5 times faster than our Trimix dives to 650 or 686 m. This shows that even Trimix 10 cannot prevent the severe h.p.n.s. initiated by this extremely rapid compression for such deep diving. In the earlier slow compression to 420 m, which caused the nitrogen narcosis, it was not that the compression was too slow but that the nitrogen content was too high for these sensitive divers. We now recommend slow (7 day) exponential compression with many 14 h stages for adaptation, and 5% (by volume) nitrogen in the Trimix.

D. H. Elliott (*Shell U.K. Limited*, *London*, *U.K.*). Among the measures that might ameliorate the effects of h.p.n.s., which Dr Bennett listed, he did not mention one that has been used with some apparent success elsewhere. Rapid compression is used to take the diver beyond his target depth and the induced h.p.n.s. is then reduced by early decompression to the target depth, at which he appears to demonstrate fewer manifestations than had he merely arrived there without the deeper excursion. What might this tell us about mechanism? What value might this approach have in practice?

P. B. Bennett. I have never used this technique, which Dr Buhlman in Zurich has found effective in reducing h.p.n.s. It requires further research.