Int Marit Health
2012; 63, 1: 49–55
www.intmarhealth.pl

ISSN 1641-9251

VIA MEDICA

ORIGINAL PAPER

Extremely deep recreational dives: the risk for carbon dioxide (CO_2) retention and high pressure neurological syndrome (HPNS)

Jacek Kot

National Centre for Hyperbaric Medicine, Institute of Maritime
and Tropical Medicine in Gdynia, Medical University of Gdansk

ABSTRACT

Clear differences between professional and recreational deep diving are disappearing, at least when taking into account the types of breathing mixtures (oxygen, nitrox, heliox, and trimix) and range of dive parameters (depth and time). Training of recreational deep divers is conducted at depths of 120–150 metres and some divers dive to 180–200 metres using the same diving techniques. Extremely deep recreational divers go to depths of more than 200 metres, at which depths the physical and chemical properties of breathing gases create some physiological restrictions already known from professional deep diving. One risk is carbon dioxide retention due to limitation of lung ventilation caused by the high density of breathing gas mixture at great depths. This effect can be amplified by the introduction of the additional work of breathing if there is significant external resistance caused by a breathing device. The other risk for deep divers is High Pressure Neurological Syndrome (HPNS) caused by a direct compression effect, presumably on the lipid component of cell membranes of the central nervous system. In deep professional diving, divers use a mixture of helium and oxygen to decrease gas density, and nitrogen is used only in some cases for decreasing the signs and symptoms of HPNS. The same approach with decreasing the nitrogen content in the breathing mixture can also be observed nowadays in deep recreational diving. Moreover, in extremely deep professional diving, hydrogen has been used successfully both for decreasing the density of the breathing gas mixture and amelioration of HPNS signs and symptoms. It is fair to assume that the use of hydrogen will be soon "re-invented" by extremely deep recreational divers. So the scope of modern diving medicine for recreational divers should be expanded also to cover these problems, which previously were assigned exclusively to professional and military divers.

(Int Marit Health 2012; 63, 1: 49–55)

Key words: recreational diving, carbon dioxide retention, high pressure neurological syndrome

INTRODUCTION

Since the beginning of recreational scuba diving, as popularized in the 1940s by Jacques Cousteau, the list of life-threatening risks for divers has included drowning, pulmonary barotrauma, and nitrogen narcosis. Soon recreational divers tried to go deeper and stay longer, so the risk for decompression sickness became evident due to the large inert gas load in the diver's tissues. Use of artificial breathing mixtures, for example nitrogen with oxygen (nitrox), helium with oxygen (heliox), and helium, nitrogen, and oxygen (trimix) to replace compressed air,

---

Jacek Kot, MD, PhD; National Centre for Hyperbaric Medicine, Institute of Maritime and Tropical Medicine in Gdynia, Medical University of Gdansk, 9B Powstania Styczniowego Str., 81-519 Gdynia, Poland; phone: +48 58 6225163, fax: +48 58 6222789; e-mail: jkot@gumed.edu.pl

and pure oxygen to enhance decompression, extended the list of risks for divers by oxygen toxicity on the central nervous system during short exposures to high partial oxygen pressure (usually exceeding 1.6 atm), and on the lungs during long exposures, even to moderately increased partial pressure of oxygen (mostly those exceeding 1.0 atm). The personal diving equipment used by recreational divers has also changed significantly. Modern deep-sea divers, sometimes called “technical divers”, use several independent breathing units (so-called “stages”), closed-circuit breathing systems, personal diving bells for long decompression, and underwater scooters, to name a few.

Interestingly, most, if not all, so-called “inventions” in recreational diving have been taken from military and professional diving. These include not only artificial breathing mixtures and pure oxygen, but also some technical solutions, like re-breathers or diving bells. Therefore, is can be expected that future trends in diving deeper for recreational purposes will be based on the techniques, procedures, and methods currently used in military and professional operations.

Until recently, professional and military diving was well differentiated from recreational diving. Professional dives were longer and deeper; they were conducted with support from the surface using diving bells, decompression chambers, hard helmets with umbilical cords supplying breathing mixtures, and closed circuit breathing systems and they were using mostly heliox and pure oxygen for decompression. Nowadays, both types of dives have similar ranges of depth and times, similar breathing mixtures used by the divers, as well as similar operational procedures - the use of oxygen for accelerating decompression is currently a standard procedure learnt during training courses for advanced divers. Technical divers uses trimix routinely when diving in the range of 50 to 150 metres, and going deeper means decreasing the content of nitrogen in the breathing mixture, so it becomes closer and closer to heliox. The use of closed-circuit breathing systems, so called “re-breathers”, is becoming more and more popular for recreation as prices for single units are getting lower. The distance travelled during cave exploration by amateurs often exceeds several kilometres, and the time spent underwater sometimes exceeds 10 hours in a single dive, so individual diving bells are used for decompression. Some individuals are known to have dived to depths of 260–300 metres solely for recreational purposes.

In fact, the only thing that still keeps professional diving different from recreational diving is the fact that professional divers receive payment for their dives, which makes them an employee [1]. As a consequence, their employer must follow the national regulations and international standards. Therefore, some steps must be taken to protect the diver including a pre-dive fit-to-dive medical examination, on-surface support with a team of staff with hyperbaric chambers, as well as restrictive procedures to be followed. The similar formal approach applies to diving instructors, as they should be recognized as a kind of professional diver, even if they follow different procedures and they do not need such on-surface support. On the other hand recreational divers, who are not rewarded for any specific dive by an employer, can conduct virtually the same dive as a professional diver (or even more risky dives due to extreme conditions), but rely on their personal free will to take any risks and to use any diving technique he or she wants or can afford.

It can be observed that recreational extreme divers are going in the same direction that professional divers followed in the past. So it is justified to expect that some solutions used to solve the physiological problems already met in professional diving will be “re-invented” in extremely deep recreation dives. These include at least two physiological phenomena, which are directly related with breathing gases under high ambient pressures, namely: increased gas density with retention of carbon dioxide (CO_2), and High Pressure Neurological Syndrome (HPNS). Both phenomena create some limits for diving deeper, especially in recreational mode, which does not have the support typically found in professional deep diving, for example low-resistance, open-circuit breathing systems with umbilical gas delivery, diving bells, and on-surface hyperbaric chambers for lengthy or unlimited diving operations.

Currently, underwater training of deep recreational divers is conducted at depths of 120–150 metres, and some divers are using the same techniques and similar procedures to dive to approximately 180 metres after their training course. Diving to such depths has inherited risk for death underwater due to oxygen toxicity, nitrogen narcosis, or intoxication with polluted gases, after surfacing due to pulmonary barotrauma as a consequence of emergency surfacing, and/or decompression illness as a consequence of inadequate decompression processes. If a diver is experienced in deep dives, is well trained with his or her personal equipment, and strictly follows procedures used for planning of such dives, the general overall risk is higher than for other types of recreational diving but quite acceptable, even for insurance companies. In this paper, only dives conducted to depths exceeding 200 metres are considered extremely deep, at least from the physiological point of view. This is because additional physiological restrictions due to high density of breathing gas and direct effect of pressure on cells start to play a crucial role. However, it must be remembered that the cut-off of 200 metres for depth is purely arbitral and does not take into account any personal factors, like capabilities to manage organisation of the expedition and mental ability to cope with the tasks overloading and consequently with the stress while underwater. Indeed, each diver has their own limit for diving and understands differentially the term "extremely deep dive".

THE AIM

The aim of this paper is a description of carbon dioxide (CO_2) retention due to increased gas density and the risk of High Pressure Neurological Syndrome (HPNS) during extremely deep (over 200 metres of depth) recreational dives.

RESULTS — DESCRIPTION OF THE RISKS

GAS DENSITY AND RETENTION OF CARBON DIOXIDE (CO_2)

The density of gas increases with pressure. This can lead to progressive restriction of total pulmonary ventilation, especially during exertion. Moreover, increased gas density may disturb intrapulmonary gas exchange because of an alteration in the regional distribution of ventilation [2].

The relative density is also directly related to the type of gas in the breathing mixture. It is possible to vary the density of the inspired gas by changing its composition. In most cases of deep dives, nitrogen is replaced with helium, which is seven times less dense. For deep, but not yet extreme, dives (up to 120–150 metres), the main reason for replacing nitrogen, or its part, with helium is the intention to limit the partial pressure of nitrogen to decrease symptoms of nitrogen narcosis. Due to the relatively high price of helium, recreational divers are introducing this gas to breathing mixture only to such an extent that the partial pressure of nitrogen is kept below some predefined level. Usually this is 3.95 atm, which is equivalent to breathing compressed air at a depth of 40 metres (79% of 5 atm). In military and professional diving, where costs are not a major issue, only heliox is used for deep dives.

An additional advantage of decreasing the content of nitrogen is a decrease in breathing gas density, and this becomes of great importance when diving deeper than 200 metres.

The density of the gas flowing in the tracheobronchial tree is one the determinants of airway resistance. An increase of ambient pressure or a change in gas composition will also affect the rate at which gases can be inhaled and exhaled. This is because the flow of gas through a tube depends also on viscosity, velocity, and the diameter of the tube [3]. All these factors define whether flow is laminar or turbulent (cut off for the Reynolds number, defined as (density × velocity × diameter)/viscosity, is 2,000). For example, the viscosity of a mixture of 79% helium and 21% oxygen (heliox) is 1.1 times that of air and the density 0.34 times that of air, so the critical velocity for transition from laminar to turbulent flow for heliox must be approximately three times that of air. Therefore, when ventilation is increased from a resting value, at first the flow of both gases will be laminar, then turbulent with air but still laminar with heliox, and finally turbulent with both gases. Practically, it means that in the transient phase increase of flow resistance for compressed air would be non-proportionally greater than for heliox.

Due to the relatively large absolute densities of nitrogen and oxygen when compared to helium (with factors of 7 and 8, respectively), there is a significant influence of those gases on density of breathing mixture when compared with helium alone. It was pointed out many years ago by Lanphier [4] that "although pure helium is less then one-seventh as dense as air, the relative density increases markedly with the addition of oxygen and nitrogen. For example, a mixture of 80% helium and 20% oxygen is almost exactly one-third as dense as air".

In order to keep the elimination of the carbon dioxide on a constant level (assuming that there is no additional work to be conducted other than diving itself) both the inspiratory and expiratory work of breathing increases significantly [5]. An excellent overview of ventilation restrictions in hyperbaric conditions is presented by Doolette and Mitchell [6]. Here, only the basic aspects related to practical issues of extremely deep dives are briefly summarized.

The work of breathing depends on external and internal factors. External factors are related to devices that allow breathing while under pressure, and internal factors are consequences of compressed gas flow through a respiratory tract.

Depending on the flow of gas inside the breathing loop, there are two kinds of underwater breathing apparatus. One is an open circuit (OC) in which the breathing mixture is delivered by the regulator from the pressure tank on demand of the diver by inspiratory effort, which is the creation of a small negative pressure (“underpressure”) in the mouthpiece. This underpressure opens the inspiratory valve and starts the inflow of the breathing mixture. After completion of inspiration, the diver’s exhalation actively opens an expiratory valve and allows free outflow of the breathing mixture into the water, counter forcing the hydrostatic pressure difference between the middle of the lungs and the level of the expiratory valve. Usually there is a kind of pressure support during inhalation, which pushes the breathing mixture into the diver’s lungs with a slight overpressure, and consequently the inhalation costs only a small additional work of breathing related with opening the valve. Therefore, in this kind of equipment the external work of breathing depends mostly on the opening pressures of two valves, inspiratory and expiratory. The negative feature of the OC is the wasting of the breathing mixture due to its exhalation into the water. It makes for inefficient use of the breathing gas, especially at very great depths, because the mass of gas delivered to achieve a normal tidal volume is directly proportional to ambient pressure (i.e. depth). Moreover, the fractional content of the breathing mixture stored in the pressure tank is fixed, so in order to keep the proper partial pressure of oxygen and nitrogen during all phases of diving, there is a need to have many different and independent gas supplies. For a depth range of 200 metres it should be at least 5 different breathing mixtures. Taking into account the need to have some backup gas supply, this means having as many as 8 to 10 pressure tanks during dives with several hours of decompression.

Alternatively, divers can use a closed-circuit system, also called a “re-breather” (CCR), which collects exhaled gas, regenerates it by eliminating carbon dioxide and adding oxygen, and redirects the gas mixture again to the mouthpiece for re-inhalation. In such devices exhaled gas flows through a loop of one-way valves, hoses, a counter-lung, and a CO_2 scrubber. This flow is generated entirely by the breathing effort of the diver, so using such a system makes the diver prone to all risks of increased work of breathing due to increased density of the breathing mixture as a result of the type of gas and increased ambient pressure. Moreover, the efficacy of the CCR heavily relies on the absorbent used for the elimination of carbon dioxide. Exhaustion of the absorbent or “channelling” along paths of uneven resistance can lead to an increase in carbon dioxide in the inspired mixture leading to life-treating hypercapnia. On the other hand, CCR uses efficient breathing mixtures and keeps the partial pressure of oxygen at a pre-set optimal level during all phases of diving. It is safe to predict that using CCR is the only method to conduct extremely deep and long dives independently from surface or diving bell support.

When analysing the internal factors of breathing underwater, it must be remembered that as pressure and gas density increases, both maximum voluntary ventilation (MVV) and maximum exercise ventilation (MEV) are progressively reduced [7]. Based on physiological measurements and mathematical calculations it has been shown that using compressed air at a depth of 150 metres (not suitable for real diving due to the risk of oxygen toxicity and significant nitrogen narcosis) limits MVV from more than 200 l/min on the surface to less than 50 l/min at depth, i.e. reducing it by a factor of four. Replacing nitrogen with helium and keeping the same fractional content of oxygen (not suitable for real diving due to the risk of oxygen toxicity) allows higher level of MVV to be attained — up to approx. 80 l/min, but this is still only one-third of the initial surface value. This restriction for the ventilation, and as a consequence the limit for the elimination of CO_2 during any additional exercise while at the bottom, is further complicated by the presence of so-called “CO_2 retainers”. This concept describes individuals who tend to retain CO_2 during exercise, and it has been described both in athletes and military divers [7]. While such internal features can be useful in normal diving, they can also lead to dramatic consequences as it puts the diver in an unfavourable situation immediately at the beginning of any emergency situation. As Lanphier [8] wrote, “Whatever its aetiology, the individual tendency to retain CO_2 during exertion appears to be the most important single factor in the problem of abnormal $PalvCO_2$ and its potentially serious consequences”. Indeed, hypercapnia by itself can lead a diver to lose consciousness [9]. Additionally, it can make divers more susceptible to oxygen toxicity (with loss of consciousness and generalized convulsions) [10], nitrogen narcosis [11], and maybe decompression sickness.

Due to difficulties with post-mortem confirmation of hypercapnia as the primary cause of death, the real rate of deaths due to CO_2 intoxication during deep dives is unknown. However, in the recent scientific literature there is at least one well-documented case of death due to carbon dioxide excess in a recreational diver diving with closed circuit breathing apparatus to an extreme depth of 264 metres [12]. Also, a report from a diver who successfully dived to 284 metres using CCR (K. Starnawski, Poland, 2011, personal communication) includes the relation of severe dyspnoea while on the bottom, probably due to increased gas density and the significant work of breathing.

This shows that there is a clear physiological limit of using what is currently the most popular breathing mixture of oxygen and helium (sometimes with a small amount of nitrogen), especially within closed-circuit re-breathers. The will to go deeper will probably stimulate recreational divers to use hydrogen instead of helium. Hydrogen is the lightest gas in nature; its density is 14 times less than nitrogen and even 2 times less than helium. Its use in professional deep dives has been successfully proven in the experiments of HYDRA 10 conducted by the French commercial company COMEX [13]. More details of physiological features of hydrogen are presented below.

HIGH PRESSURE NEUROLOGICAL SYNDROME (HPNS)

High Pressure Neurological Syndrome (HPNS) occurs during fast compressions to great pressure, usually exceeding 15 atm (150 metres). Signs and symptoms include tremor of the extremities, severe myoclonic jerks, fatigue, imbalance, incoordination, and — ultimately — convulsions. Those symptoms can be accompanied by changes in the electrical activity of the brain, such as nausea, dizziness, and somnolence. Further compression can lead to death [14].

The development of HPNS is affected by the rate of compression and the hydrostatic pressure attained, so the faster the rate of compression and the higher the pressure, the more severe the signs and symptoms. This becomes an increasingly important consideration during extremely deep recreational dives when divers use as fast compression rates as possible to avoid unnecessary saturation with inert gas which could prolong decompression. Such an approach works fine for most bounce dives in the range of 150 metres, but during extreme deep dives this approach enhances symptoms of HPNS. Indeed, many divers diving to depths greater than 200 metres report tremors of extremities which limit their intentional movement. This can be of great importance during any emergency situation, when divers should be able to take fast decisions and carry out precise operation of equipment.

On the other hand, decreasing the compression rate usually alleviates signs and symptoms of HPNS. Such observations are well documented from saturation exposures lasting many days to pressures exceeding 30 atm (300 metres). Unfortunately, in order to avoid HPNS symptoms on arrival to such depths, the compression rate must be extremely low, and even then some stages of constant pressure for adaptation are usually required. For example, when exposing divers to the pressure equivalent to 366 metres during a study dive in Philadelphia [15], it was possible to avoid HPNS when the compression rate was only 1.5 m/min between 122 and 274 metres, then it was even slowed to 0.8 m/min between 274 and 366 metres, and additionally there were some stages of two to four days included at 122, 213, and 274 metres. For the future challenge of even deeper diving, it must be remembered that when diving beyond 330 metres HPNS may be present regardless of the compression rate [16].

Recreational deep divers try to solve the problems with HPNS caused by fast compression by using even faster compression and then fast ascending to the first decompression stop in order to shorten the time of exposure and to avoid the outbreak of HPNS signs and symptoms. The compression rate of 5 to even 15 m/min was used with success before the 1980s, and a test dive with compression to 180 metres over a 15-min period with a total duration of two hours has been advocated for the prediction of a diver's sensitivity to higher pressures [17]. This test has been successfully validated by monitoring the theta activities in EEGs in the frontal area of the scalp both during the test dive to 180 metres and then to 450 metres [18].

The other methods to suppress HPNS signs and symptoms [19] include: selection of the least susceptible divers; choice of a suitable rate of compression involving an exponential profile with stages during the compression; excursions from saturation at a shallow depth; the use of anaesthetics such as nitrogen added to the heliox to produce a so-called trimix (helium — nitrogen — oxygen); and other factors such as allowing time for adaptation after compression before starting work. Unfortunately, most of them, with the exception of using nitrogen (or another anaesthetic gas) cannot be applied in recreational diving.

It is generally assumed that the majority of HPNS symptoms are mostly caused by the compression effect, presumably in the lipid component of cell membranes of the central nervous system. This effect was firstly noted during deep dives with gas low lipid solubility (helium), which allowed high hydrostatic pressures to be applied to humans. The compression effect in the lipid component of the membrane can be counteracted by using a gas with sufficient solubility in the breathing mixture, including anaesthetic gas or nitrogen, or even hydrogen [14].

Hamilton reported [20] the successful use of a compression rate of 30 m/min up to a depth of 305 metres with trimix containing 13% nitrogen during the ACCESS series. Rostain published the results [21] of the CORAZ series in which they compared trimix with different fractional contents of nitrogen: 0, 4.5, and 9% during three dives to 300 metres with a total compression time of 4 hours. Trimix with 4.5% nitrogen appeared to be the best overall for ameliorating HPNS without causing significant euphoria due to the narcotic properties of nitrogen (“nitrogen narcosis”). In an anecdotal report, Krasberg claimed in 1979 that he performed a “typical oil field task” at a depth of 305 metres after 35 minutes of compression with a 6.4% nitrogen plus 1 1/2 oz Scotch whiskey (after [22]). A similar fractional content of nitrogen in trimix (5–10%) has also been used in the ATLANTIS series of chamber saturation exposures to 650 and 686 metres [23].

The use of hydrogen to ameliorate symptoms of HPNS was tested by the French company COMEX during their HYDRA experiments, with final exposure with hydrogen — helium — nitrogen — oxygen (27%, 65%, 7%, and 0.56%, respectively) mixture to 701 metres [13, 24]. It was the saturation in-chamber exposition which took in total 42 days, including 15 days for compression alone. The parameters of this exposition are not applicable for recreational deep dives, but the HYDRA series clearly showed several important features of using hydrogen as a useful gas in breathing mixtures for deep divers: it can be used safely without a significant fire risk when the content of oxygen is kept below 4–5%; there were some “narcotic problems” reported by divers, which exclude its use as the only inert gas in the breathing mixture; it reduces or suppresses neurological symptoms of HPNS, including tremors; and its low density reduces work of breathing. Hydrogen has also been used for deep bounce dives in the 1940s by Arne Zetterström in the HYDROX series of dives to 160 metres using a breathing mixture of 96% hydrogen and 4% oxygen [25]. The death of Zetterström during this dive was related to miscommunication with the surface support team and — in consequence — too rapid surfacing, and not with breathing the experimental breathing mixture.

Theoretically, any anaesthetic gas which expands the lipid component of the central nervous system cell membrane will ameliorate symptoms of HPNS. Several gases other than nitrogen have been tested, at least in animal models, namely argon, nitrous oxide, carbon tetrafluoride, and sulphur hexafluoride [26].

The optimal dose of the anaesthetic gas has not been yet defined, but using only the minimal assumptions for a biochemical model of pressure counteracting with a narcotic gas in the two-dimensional matrix of the biological membranes, it has been calculated for example for dives of 300 metres (31 ata, 1000 fsw) that for about every 9 atm helium, 1 atm nitrogen would be required for prevention of HPNS [27]. This result is in good agreement with experiments with humans when 10% nitrogen was successfully added to heliox for amelioration of HPNS symptoms. Extrapolation of these calculations for other gases allows the prediction that, for example, 0.5% of nitrous oxide (a well-known anaesthetic gas, known also as “laughing gas” or “sweet air”) should have a similar effect, and indeed this was confirmed in an animal model.

It is unknown whether in future hydrogen will be used routinely by recreational divers for extremely deep dives, due to its flammability. But from the physiological perspective its use solves both physiological restrictions of extremely deep exposures by decreasing breathing mixture density, and ameliorating HPNS symptoms. So it is justified to predict that this gas will soon be “reinvented” by extremely deep recreational divers.

CONCLUSIONS

When diving to extreme depths, exceeding 200 metres, recreational divers are prone to risks known already from professional diving, including carbon dioxide retention due to increased gas density and High Pressure Neurological Syndrome. So it should be expected that the scope of modern diving medicine for recreational divers should be expanded also by these subjects, which previously were assigned exclusively to professional and military divers.

REFERENCES

1. Elliott D. The Foundation of our Future. Invited lecture. The 37th Annual Meeting of the EUBS, 24th–27th August 2011, Gdansk, Poland.
2. Wood LDH, Bryan AC. Mechanical limitations of exercise ventilation at increased ambient pressure. In: Lamberts-

en CJ (ed.). Undersea physiology. Proceedings of the Fourth Symposium on Underwater Physiology. Academic Press, New York 1971.

3. Murphy TM, Clark WH, Buckingham IP, Young WA. Respiratory gas exchange in exercise during helium-oxygen breathing. J Appl Physiol. 1969; 26: 303–307.
4. Lanphier EH. Interactions of factors limiting performance at high pressures. In: Lambertsen CJ. Underwater physiology. Proceedings of the Third Symposium on Underwater Physiology: 375–385. Williams and Wilkins, Baltimore 1967.
5. Wood LDH, Bryan AC. Effect of increased ambient pressure on flow volume curve of the lung. J Appl Physiol 1969; 27: 4–8.
6. Doolette DJ, Mitchell SJ. Hyperbaric conditions. In: Comprehensive physiology. Published Online: 1 November 2010. DOI: 10.1002/cphy.c091004. John Wiley and Sons, Inc. Online ISBN: 9780470650714.
7. Camporesi EM, Bosco G. Ventilation, gas exchange and exercise under pressure. In: Brubakk AO, Nauman TS (eds.). The Bennett and Elliott's physiology and medicine of diving. 5th edition. Saunders 2003: 77–114.
8. Lanphier EH. Pulmonary function. In: Bennett PB, Elliott DH (eds.). The physiology and medicine of diving and compressed air work. 2nd edition. Bailliere Tindall, London 1975: 102–154.
9. Morrison JB, Florio JT, Butt WS. Observations after loss of consciousness underwater. Undersea Biomed Res 1978; 5: 179–187.
10. Clark JM, Gelfand R, Lambertsen CJ, Stevens WC, Beck G Jr, Fisher DG. Human tolerances and physiological responses to exercise while breathing oxygen at 2.0 atm abs. Aviat Space Environ Med 1995; 66: 336–345.
11. Hesser CM, Adolfson J, Fagraeus L. Role of CO_2 in compressed air narcosis. Aerospace Med 1971; 42: 163–168.
12. Mitchell SJ, Cronjé FJ, Meintjes WA, Britz HC. Fatal respiratory failure during a "technical" rebreather dive at extreme pressure. Aviat Space Environ Med 2007; 78: 81–86.
13. Gardette B, Massimelli JY, Comet M, Gortan C, Delauze HG. Deep hydrogen diving: HYDRA 10 — a 701 msw record dive. Abstract of the Undersea and Hyperbaric Medical Society, Inc. Annual Scientific Meeting held July 7–10, 1993.
14. Bennett PB. High pressure nervous syndrome. In: Schilling CW, Carlston CB, Mathias RA (eds.). The physicians's guide to diving medicine. Plenum Press, New York and London 1984: 109–127.
15. Lambertsen C.J. Predictive studies. IV. Physiological effects and work capabilities in He-O2 excursions staged to pressures of 400–800–1200 and 1600 fsw. In: Proceedings of the Symposium: The Working Diver. Washington DC: The Marine Technology Society1976: 156–173.
16. U.S. Navy Diving Manual, Revision 6, SS521-AG-PRO-010, 0910-LP-106-0957, 15 April 2008.
17. Fructus X. Limits for both open water and chamber diving. In: Smith EB, Halsley MJ, Daniels S (eds.). Techniques for diving deeper than 1,500 feet. UHMS Workshop, Wilmington, North Carolina, USA 1980: 27–35.
18. Rostain JC, Lemaire C, Gardette-Chauffour MC, Doucet J, Naquet R. Estimation of human susceptibility to the high-pressure nervous syndrome. J Appl Physiol. 1983; 54: 1063–1070.
19. Halsey MJ, Settle W, Smith EB. Strategy for Future Diving to Depths Greater than 1,000 Feet. UHMS Workshop, 1975.
20. Hamilton RW, Schmidt TC, Kenyon DJ et al. ACCESS: diver performance and physiology in rapid compression to 31 ats. Tech Memo CRL-T-789. Tarrytown, Union Carbide Co, New York 1974.
21. Rostain JC, Gardette-Chauffour MC, Naquet R. HPNS during rapid compressions of men breathing He-O2 and He-N2-O2 at 300 m and 180 m. Undersea Biomed Res 1980: 7: 77–94.
22. Bennett PB, Rostain JC. The High Pressure Nervous Syndrome. In: Brubakk AO, Nauman TS (eds.). The Bennett and Elliott's physiology and medicine of diving. 5th edition. Saunders 2003: 323–357.
23. Bennett PB. To the very depths: a memoir of Professor Peter B Bennett. Best Publishing Company 2008.
24. Lafay V, Barthelemy P, Comet B, Frances Y, Jammes Y. ECG changes during the experimental human dive HYDRA 10 (71 atm/7,200 kPa). Undersea Hyperbaric Med 1995; 22: 51–60.
25. Ornhagen H. Hydrogen-Oxygen (Hydrox) breathing at 1.3 MPa. National Defence Research Institute. FOA Rapport C58015-H1. 1984.
26. Smith RA, Dodson BA, Miller KW. The interactions between pressure and anaesthetics. Philos Trans R Soc Lond B Biol Sci 1984; 304: 69–84.
27. Simon S, Katz Y, Bennett PB. Calculation of the percentage of a narcotic gas to permit abolition of the high pressure nervous syndrome. Undersea Biomed Res 1975; 2: 299–303.