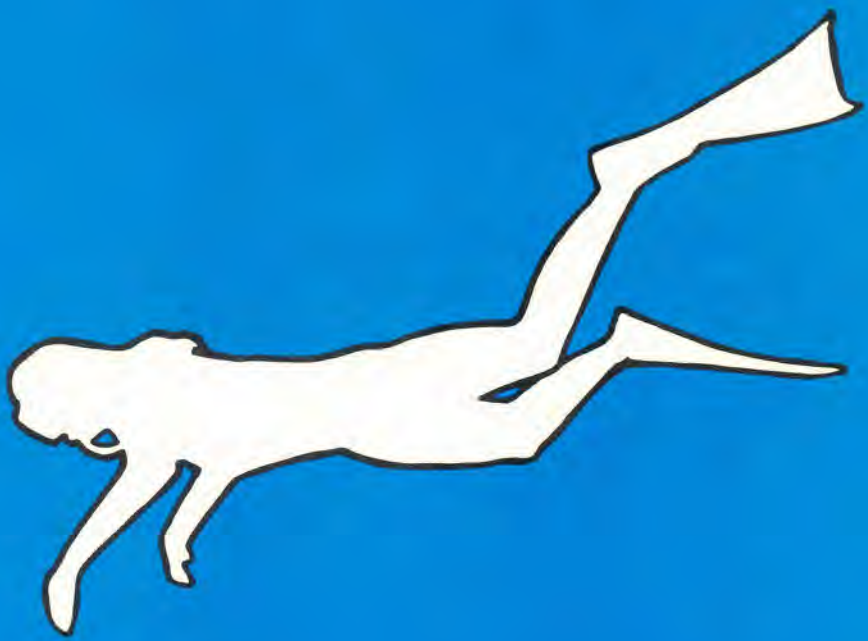
OXYGEN AND THE DIVER



KENNETH DONALD

OXYGEN AND THE DIVER

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OXYGEN AND THE DIVER

KENNETH DONALD

This book is the first to give a comprehensive review of the dangers to the diver when breathing oxygen.

Professor Donald, who is the accepted authority in this field, describes how unexpected hazards attended the sudden expansion of self-contained free diving during the relentless underwater combat of the Second World War. This is the only full account of the unique series of human experiments in which the safe times and depths for oxygen diving were established.

The author has reviewed all other important studies of oxygen toxicity in divers up to the present day. Considerable care has been taken to determine the original details of these investigations, many of which have not been fully published.

After the dangers of oxygen had been demonstrated during the 1940s, the almost universal use of air was adopted for shallow water swimming and diving up to fifty metres. It has since been supplemented by oxygen-nitrogen mixtures. Mixture diving is more economical of gas and allows the diver to go considerably deeper without the risk of oxygen poisoning or need for staged decompression. Much emphasis has been placed in the book on the dangers of the imprudent use of mixtures. Other factors which may affect the incidence of oxygen poisoning are also reviewed.

Whilst the book is written primarily for diving experts, many of the diving fraternity will find it an enjoyable and invaluable source of reference.

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AUTHOR

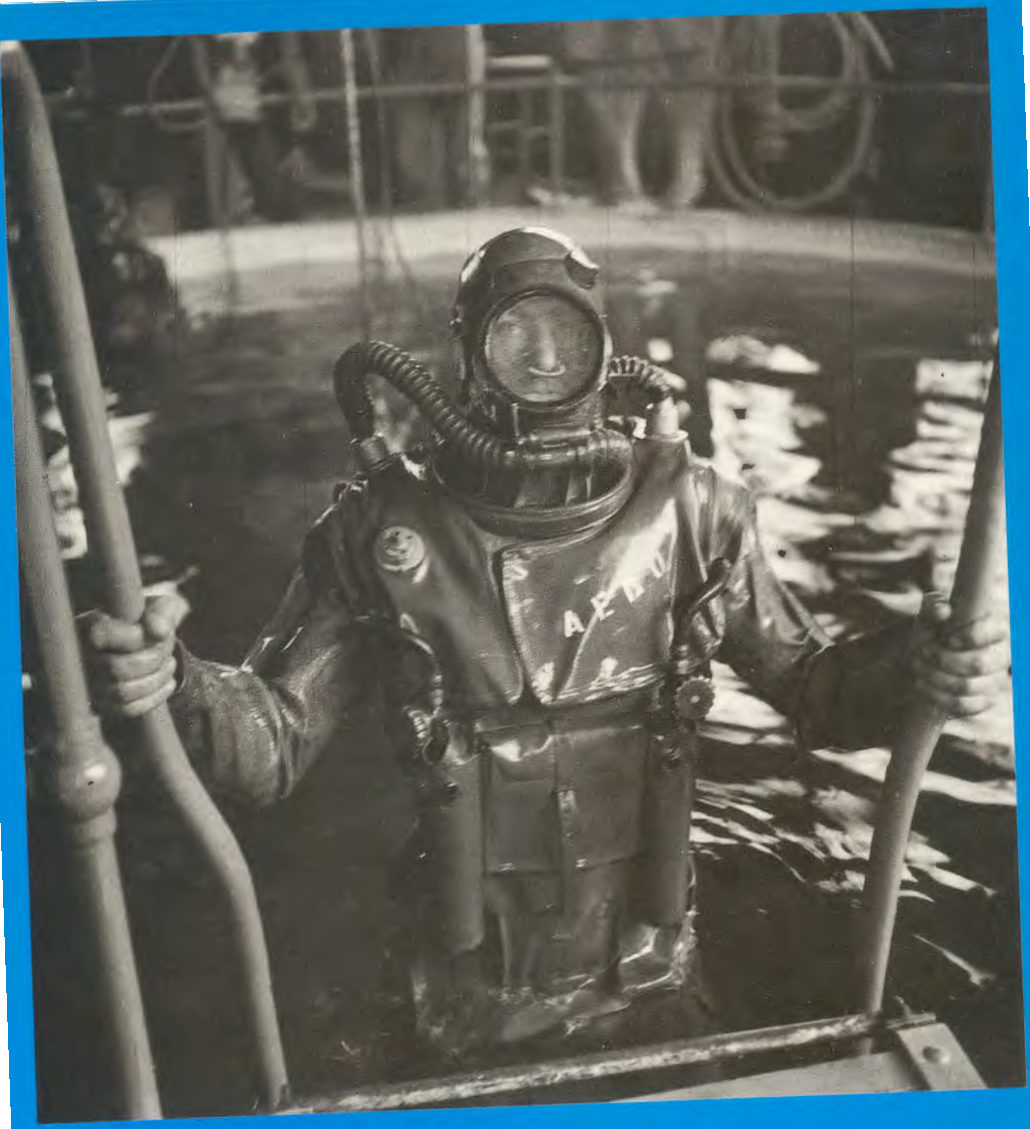
Professor Ken Donald entered the Royal Navy in 1939, having established himself as an outstanding scholar and junior doctor at Emmanuel College, Cambridge, and St Bartholemew's Hospital, London. He served as Flotilla Medical Officer in HMS Hotspur and earned a Distinguished Service Cross at the first Battle of Narvik. He and his wounded finally escaped via the Lofoten Islands.

In 1942 he commenced his famous studies into the toxic effects of hyperbaric oxygen.

After the war he returned to academic clinical medicine, initially at St Bartholemew's Hospital and later at many other famous hospitals, culminating in the Chair of Medicine at Edinburgh University in 1959. His clinical research, particularly into heart and lung disease, has achieved worldwide recognition.

Professor Donald has always maintained close links with the Royal Navy, with particular interest in diving, submarine and survival medicine. He became Chairman of the Underwater Physiology Sub-Committee (RNPRC) in 1954 and the Chairman of the main Royal Naval Personnel Research Committee in 1969. He has also served, since 1975, as Chairman of the Advisory Committee to the Secretary of State for Scotland on the safety of divers in the North Sea Oil Industry.

Professor Donald, who admits to being "something slightly over 65", now lives with his wife on the beautiful Welsh borders.



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Dedication

To Rethe Pearl

Acknowledgements

Firstly to those who helped me in the large oxygen poisoning study described in Chapter 2. These are detailed at the end of that chapter.

Secondly I owe a great deal to members and officers of the Royal Naval Personnel Research Committee and its various subcommittees and working parties, many of which I had the honour of chairing. I am particularly grateful to Frank Smith and Jimmy Hamlyn of the secretarial staff.

I have appreciated the help and support of friends and colleagues in the Royal Naval Medical School and the Royal Naval Physiological Laboratory. I would particularly like to thank Dr. Reginald Withey for his encouragement and help. I am grateful for permission to use Admiralty photographs.

Finally I would like to express my gratitude to all the experimental divers I have worked with over the years. Their cheerful companionship, trust and courage have lightened the onerous task of exploring and defining the dangers of diving.

Royal Navy Frogman (oxygen breathing), 1943. Note high degree of stream-lining of suit, cylinders, weights and counterlung (upper chest and shoulder). Note division of counterlung (inspiratory and expiratory) with canister between at back.

PREFACE

OXYGEN AND THE DIVER

The purpose of this monograph is to provide a useful, accurate and critical account of previous studies of oxygen poisoning in relation to diving. It is not an historical treatise but rather an attempt to remedy the remarkable dearth of publications of the original objective data on, or related to oxygen poisoning under water. All the material presented is immediately relevant to present day oxygen or hyperbaric diving. It is hoped that those interested in diving will find this book a convenient source of reference to previously scattered and often inaccessible reports.

Oxygen poisoning is, after decompression sickness, the second great danger in diving, a danger that will increase with the increased use of oxygen-rich gas mixture in various forms of diving. Yet, astonishingly, there is no monograph devoted to the subject.

It is sometimes remarked that, as long as the diver and his supervisor stick to the rules and regulations, they do not need to know the complex details and the full 'horror' of acute oxygen poisoning. Yet no-one makes this suggestion with regard to the causation and the varying clinical pictures of decompression sickness.

The rules concerning both air and hyperbaric oxygen diving are, on occasions, inevitably stretched or broken in the almost infinitely variable world-wide scenarios of diving. There can be no excuse for not extending this open sophisticated attitude with regard to decompression sickness to all potential risks to the diver, including oxygen poisoning. The greater the knowledge of these dangers the greater the safety of the diver.

Oxygen poisoning under water did not arise as a problem until the second world war when independent divers began to breathe pure oxygen during covert operations. A large number of studies were carried out by the Royal Navy, mostly under water. It is very doubtful whether

experimental diving on oxygen of large groups to 'acute toxic end-points' will ever be undertaken again and, for this reason, the results, which cover many past and present problems are made fully available for the first time.

The author then describes a series of studies of oxygen poisoning, mainly unpublished, by the United States Navy. I am most grateful to them for providing copies of many original U.S.N. reports. The initial apparent difference of sensitivity to oxygen poisoning in the two navies is discussed. Butler & Thalmann (U.S.N.) and their colleagues also provided me with a great deal of material concerning their recent research into prolonged oxygen exposures at shallow depths, with deeper excursions.

Finally the present practises in oxygen diving are described and possible procedures or regulations to further reduce the hazards of oxygen poisoning are proposed.

In chapter 4 the risks of lung damage in divers due to oxygen toxicity are discussed. An even more cautious attitude to this problem is recommended.

The use of hyperbaric oxygen to accelerate the elimination of inert gases during decompression after air or oxy-helium diving is also reviewed. The particular dangers of underwater oxygen breathing decompression stops are emphasised.

Chapter 5 describes the development of oxygen-nitrogen mixtures in counter-lung self contained diving. These mixtures are being increasingly used by service and commercial divers. Several new types of oxygen-nitrogen mixture breathing apparatus have been introduced and the advantages and disadvantages of such apparatus are discussed, particularly in relation to oxygen poisoning. The important claim that oxygen toxicity is greatly enhanced by hyperbaric nitrogen when breathing oxygen-nitrogen mixtures and the resulting restriction of acceptable oxygen partial pressure allowed in such mixture diving, is examined. The supporting evidence of this claim, previously unpublished, is presented and reviewed.

In chapter 6 problems in relation to carbon dioxide and hyperoxic diving are discussed. The first occurrence of carbon dioxide narcosis in oxygen underwater swimmers is described. The widely accepted claim that under-ventilation and carbon dioxide retention occur in oxygen-nitrogen mixture divers with the enhanced risk of oxygen toxicity is discussed. An

abstract of Lanphier's large unpublished series of ventilation and end-tidal PCO_2 measurements in exercising divers on oxygen, air and oxygen-nitrogen mixture at various depths is presented and reviewed in some detail. The associated studies of the ventilatory response to inhaled carbon dioxide and to exercise at atmospheric pressure, breathing air, in a group of so-called 'CO_2 retaining' mixture divers is also reported.

In chapter 7, titled 'Are divers really different?', a general discussion is followed by a detailed review of many important studies purporting to show that divers have an abnormally low ventilatory reaction to exercise and to increased levels of carbon dioxide.

After examining the evidence in chapters 6 and 7 it is proposed that far more direct and convincing evidence is required to support the hypotheses that divers, in general, have abnormal ventilatory behaviour and that mixture divers, in particular, are even more vulnerable to oxygen poisoning.

Finally, those interested in the naval events related to this research may find it useful to refer to Appendix 1 before reading Chapter 2.

CHAPTER 1

ADVERSE EFFECTS OF OXYGEN AT INCREASED PRESSURES

Man, in order to remain under water, is forced to employ artificial aids and breathe gases at tensions which are never encountered in the natural environment. There is no collapse of the lungs which continue to ventilate with the gases supplied to them at that pressure. The pulmonary and general circulation are scarcely altered. In these conditions it is inevitable that the blood will equilibrate with the high tension of gases in the alveoli and that the tissues throughout the body will absorb and contain gases at these tensions. It is difficult to suggest on theoretical grounds what the reactions of living matter to high oxygen tensions will be. The protoplasm of any living organism has behind it and within its substance a long heritage of successful evolution and adaptation. Heat, cold, hypoxia, starvation and disease have been encountered and successfully resisted again and again. If an organism is exposed to such hazards it is logical to expect processes and patterns of physiological behaviour to meet these emergencies, even if some are atavistic and no longer fully effective. Living matter is wise and old and experienced but it is doubtful if it has ever encountered such tensions of oxygen.

It has long been a matter of debate why deep diving mammals such as the whale do not appear to suffer from oxygen poisoning or decompression sickness. Whales normally sound to about 300 fsw but have been known to dive to depths as great as 3000 fsw in 'escape' dives after harpooning. However, when diving, the lungs and thorax, which are capable of a far greater degree of volume reduction than in the land mammal, transfer much of the compressed air to the large dead space. In any case, even if the lung gases were fully absorbed, one single lungful is quite incapable of raising the gas content and tensions in the whale's body tissues to any

important or harmful degree. The whale's respiratory adaptations include the extreme mobility of the chest wall, allowing dives far below the limited range of breath-holding man, and marked tolerance of intermittent hypoxia and hypercarbia. It is the use of elaborate artifacts by man, which allow the continuation of normal respiration and the continued absorption of gases at increased pressures, that cause these unnatural diseases.

Thus it appears that the tissues of man breathing pure oxygen or air at pressure are exposed to an internal environment which has been previously unknown to living matter. A number of savants remarked on the possible toxic action of oxygen at increased concentrations shortly after its discovery in the atmosphere and the promulgation of the combustion theory of respiration. They claimed that the respiratory exchange would be increased, the circulation accelerated and the lungs congested and that even inflammation and death would occur. Lavoisier and Seguin (1789) denied such an increase in the oxidative processes of the body. Regnault and Reiset (1849), in a series of experiments, demonstrated that animals, exposed to an atmosphere rich in oxygen, showed no convincing evidence of any increase in their oxidative processes.

Paul Bert (1878) made enormous strides in our knowledge of the effect of varying tensions of gases. His brilliant work has withstood the test of time in the most impressive manner. He showed that oxygen at increased pressure was highly poisonous and that no living matter was exempt. Larks exposed to 15 to 20 atmospheres of air convulsed and finally died. A similar effect was obtained in these birds in approximately one fifth of this pressure of oxygen. In a large series of experiments, with many species, Bert showed that the oxygen tension was the decisive factor in the immediate effect of air or any mixture of nitrogen and oxygen at pressure. Nitrogen was apparently inactive except in its role in bubble formation with decreasing pressure. He suspected that the convulsions were caused by the action of a secondarily produced poison on the central nervous system. He therefore transfused normal animals with large quantities of blood from those that had been severely convulsed with oxygen. His results were entirely negative and he concluded that the toxic action of oxygen was a direct one. Bert demonstrated that, although oxygen was a general poison, the central nervous system was the first to be so grossly affected that it caused violent convulsions and death before marked

changes in other organs could be demonstrated. In this context he showed that the mesodermal tissues of animals, killed in this way, could be grafted and survive in healthy animals. Bert did not consider his experiments sufficiently controlled, especially with regard to the carbon dioxide present, to arrive at definite conclusions concerning the variation of tolerance in different species. He made the important observation that, contrary to all previous proposals, high partial pressure of oxygen caused an actual diminution in oxidation and a fall in body temperature. He also demonstrated that high oxygen tensions had an adverse effect on insects, arachnids, myriapods, molluscs, earthworms, fungi and germinating seeds. It inhibited the putrefaction of meat and delayed the souring of milk. He demonstrated, in other words, that oxygen at increased tensions acted as a general protoplasmic poison. He attempted to determine the most 'favourable' tensions of oxygen for metabolic activity and concluded 'that increase of oxygen tension above its normal value in ordinary air seemed to bring no advantage, far from it. When any difference is noticeable it is in favour of normal air.' It should be noted that in a number of his experiments, particularly with larger animals, the tensions of carbon dioxide allowed to develop were very high and the decompression was far too rapid. It is possible that they suffered from carbon dioxide poisoning as well as oxygen poisoning and even decompression sickness and gas embolism after decompression. Many subsequent investigators have demonstrated the convulsions of oxygen poisoning in various species (Lorrain Smith (1899), Hill and Macleod (1903), Argyll-Campbell (1929), Shilling and Adams (1933), Libbrecht and Massart (1937), Bean and Rottschafer (1939), Behnke et al (1934), but only brief mention will be made of references which may have some relevance to the present diving study.

Shilling and Adams (1933) studied acute oxygen poisoning at high pressure in large groups of rats. Although they mentioned the extreme individual variation of oxygen tolerance and the frequent occurrence of violent convulsions without any warning signs, they wrote, with regard to oxygen poisoning in man, "From our observations on animals we are of the opinion that man exposed to dangerous tensions of oxygen will have early and ample warning of convulsive seizures." Brown, Dickens and Feldberg (personal communication, 1943) carried out experiments to determine whether increased oxygen pressure would affect the mechanism of

synthesis or release of acetylcholine in sliced or ground brain tissue. No difference could be detected in the extent of acetylcholine synthesis when the brain tissue was incubated in air, or in oxygen, at pressures of 4 to 5 atmospheres. Gersh (1944) carried out extensive investigations of oxygen poisoning in cats. He convulsed these animals daily, but relieved the pressure immediately convulsions commenced and allowed the animals to recover. Complete recovery occurred in a short time and there were no neurological findings after one convulsion. These animals showed increased tolerance to oxygen at high pressure as the series progressed. After a week of daily exposures the animals began to show definite signs of neurological damage. Hopping, placing and dropping reactions were lost, especially in the forelegs. These reflexes are mediated by the cortico-spinal tracts. Increased extensor tone was also noted. A series of longer exposures at lower pressures caused more damage than brief exposures at higher pressures. After a rest of several weeks the animals showed complete recovery and their tolerance returned to the original levels. Thus it appears that the increase in 'tolerance' after repeated exposure is probably caused by cumulative damage of the more sensitive 'trigger zones.' The recovery reported is reassuring but behavioural tests were not done.

There is another important aspect of oxygen intoxication of the central nervous system. This is the apparent impairment of respiratory control (Bean and Rottschaffer, 1939). These respiratory changes may herald the onset of acute oxygen poisoning. There is usually a preliminary stage of hyperpnoea with jerky, irregular respiration which may change into slow, deep and laboured breathing. Expiratory effort becomes progressively pronounced and prolonged. Apnoeic periods may appear in the inspiratory phase and last for well over a minute. In describing these changes, the authors remarked upon the great variation in the degrees and type of respiratory disturbance.

Pulmonary Toxicity. Reverting to the end of the nineteenth century, Lorrain Smith (1899) demonstrated, for the first time, that animals breathing oxygen at moderately high tensions over prolonged periods suffered pulmonary damage. He showed that mice, exposed to 70 to 80% of an atmosphere for four days, developed congestion and consolidation of the lungs that was usually fatal. Higher tensions caused the onset of these events in the lungs in a much shorter time. As regards pathological

changes, Smith stated "Tissues of the lungs showed intense congestion in the large and small blood vessels. The alveoli were, to a large extent, filled with exudate, which was granular and fibrillated in appearance, but did not give the fibrin stain by Weigert's method, nor with eosin." No increased leucocytes or bacteria were found. Mice exposed to 3.5 atmospheres of oxygen died from pulmonary damage in five hours. They showed no convulsive symptoms although larks convulsed at this pressure in a few minutes. Many species of animals gave very similar tolerance times as regards pulmonary pathology. Smith pointed out that the lung epithelium was directly exposed to the high tension of oxygen and that the internal biochemical defences could afford it no protection. At 4.5 atmospheres some animals convulsed and others died of pulmonary damage. Smith noted the striking reversibility of this lung damage. He stated "It was found that mice have remarkable powers of recovery from the effects of high oxygen tensions. The same is probably true of man, so that in alternation between ordinary atmosphere and the atmosphere where oxygen is at an increased tension, there would be much less danger than in an exposure which is continuous."

Stadie, Riggs and Haugaard (1944), in their review, quite unaccountably belittled Lorrain Smith's outstanding contribution. French scientists, however, pay due compliment to these two great pioneers by describing the convulsive action of hyperbaric oxygen as 'L'effet Paul Bert' and the damaging effect upon the lung as 'L'effet Lorrain Smith.' Smith's findings were amply confirmed by many other workers. Some studies are of particular interest in the present context. Hill and Macleod, (1903) noted the considerable variation of individual resistance to pulmonary damage. They also produced similar lung pathology by exposure to 7 to 10 atmospheres of air (1.47 to 2.00 atmospheres of oxygen) in dogs, cats and mice. Barach (1926 showed that 80 to 85% I ATS of oxygen caused fatal pneumonia and that in one rabbit, inhalation of 70% oxygen for twelve days was followed by pulmonary oedema. He attempted to acclimatise these animals by gradually increasing the oxygen from safe concentrations up to 80 to 85% of 1 ATS. In all instances there was no acclimatisation and death resulted. He concluded that the highest concentration compatible with safety was 60% of 1 ATS.

Faulkner and Binger (1927) showed that oxygen (95% 1 ATS) had no effect on frogs nor on turtles unless they are warmed to 37.5°C when

pulmonary changes similar to those found in mammals occurred.

Temperature. Bert (1878) and Hill & Macleod (1903) reported a marked fall in body temperature in various species with acute oxygen poisoning. There were no controls in either study. Bert also demonstrated the remarkable resistance of cold blooded animals to the convulsant effect of hyperbaric oxygen. Gersh (1944) found in animal experiments that a very wide variation of ambient temperature (4 to 41°C) made no significant difference to the rate of onset of oxygen poisoning at 8 ATA. He did not confirm the previously reported fall of temperature with toxic exposures to hyperbaric oxygen.

Tissue Enzymes. Only brief reference will be made to this subject. Paul Bert (1878), with his usual prescience, proposed that inactivation of the tissue enzymes occurred in oxygen poisoning. He demonstrated that a strip of living beef muscle showed diminished oxygen uptake and carbon dioxide production at high tensions of oxygen as compared with control experiments in compressed air. He wrote "Now it is a fact of the highest interest that in the presence of this free oxygen which is simply dissolved, inner oxidations slow up, then stop . . . I know nothing in physiological chemistry more curious than this effect of dissolved oxygen." Massart (1936), Libbrecht & Massart (1937), Bohr & Bean (1940), Quastel (1939) and Dickens (1945) and many others have all confirmed Bert's findings and have demonstrated tissue enzyme deactivation.

Oxygen Poisoning in Man. The first recorded exposure to hyperbaric oxygen, apart from that occurring in air diving, was by Bornstein (1910). Two engineers breathed 90 to 95% oxygen for 30 minutes at 3 ATA in the Elbe Tunnel. Bornstein breathed oxygen under the same conditions for 48 minutes. None had untoward symptoms. In 1912 he breathed oxygen at the same pressure while exercising on a bicycle ergometer. He ceased oxygen breathing after 51 minutes after suffering cramps in the hands and legs from which he soon recovered (Bornstein & Stroink, 1912).

In 1930 during investigations carried out by the Admiralty Committee on Deep Diving, twelve subjects were subjected to two atmospheres absolute of pure oxygen for one hour and four subjects to three atmospheres absolute for 30 minutes, without untoward effects (dry pressure chamber). These air diving trials were up to 325 fsw, where partial pressures of oxygen of 1.7 to 2.27 ATA were encountered. The divers showed confusion and amnesia as well as 'unreliable' and unpredictable behaviour (Damant, 1930).

J.S. Haldane (père) thought that these dangerous symptoms were due to the raised tensions of oxygen despite the lack of supporting evidence in the oxygen exposures mentioned above (Haldane & Priestley, 1935). Hill & Phillips (1932) attempted to explain the findings on psychological grounds. There is more than a hint of unreality in their explanation and discussion of the psychological peccadilloes of a group of specially selected deep sea divers of the Royal Navy. Behnke, Thomson and Motley U.S. Navy (1935) first advanced the theory, later confirmed, that the increased tension of nitrogen was the cause of these mental disturbances.

In 1933 two British naval officers, Damant and Phillips, breathed oxygen in a compressed air chamber at four atmospheres absolute. Leonard Hill was in attendance. Convulsive symptoms occurred in 16 and 13 minutes respectively. In the first case, (Damant), violent twitching of the face was experienced which was immediately relieved by reverting to air breathing at pressure. In the second case, the subject also reverted to air breathing at the same pressure after twitching of the lips had occurred. Despite this he convulsed. These findings were not published until two years later (Thomson, 1935). Stadie, Riggs and Haugaard in their review (1944) appeared to be under the impression that this officer convulsed while returning to normal pressure and suggested that air embolism could not be excluded. This is incorrect as the subject convulsed at four atmospheres of air after removing his mouthpiece. He was under the direct observation of Sir Leonard Hill throughout (personal communications, 1943, Hill and Thomson).

Behnke, Johnson, Poppen and Motley (1935) carried out a series of observations on human subjects breathing oxygen at one to four atmospheres absolute. The subjects were seated. Blood pressure and spirometric recordings were made and blood changes studied.

One Atmosphere: 10 subjects breathed oxygen for four hours. Nine were completely unaffected. One showed slight rise in blood pressure and pulse rate in fourth hour.

Two Atmospheres (abs): 3 subjects breathed oxygen for three hours. There was no subjective change, blood pressure change or increase in leucocyte counts.

Three Atmospheres (abs): 4 subjects breathed oxygen for three hours. The only change was a slight increase in leucocyte count.

Four Atmospheres (abs): 2 subjects breathed oxygen. The first subject suffered acute syncope after 43 minutes, which was relieved by reverting to air. The second subject had twitching of left eyebrow, after 44 minutes, and then gave a sudden cry and convulsed.

In 1936 Behnke, Forbes and Motley carried out further hyperbaric oxygen exposures. Four men breathed oxygen at 3 atmospheres absolute for 3 to 4 hours. All tolerated oxygen well for three hours but showed marked facial pallor, dilatation of the pupils, rise of the diastolic blood pressure of about 10 mm Hg and impairment of visual acuity, up to 25%, towards the end of the period. There were no abnormal subjective symptoms. In the fourth hour three subjects suffered abrupt onset of vertigo, nausea and a sensation of impending collapse. When turned onto air they showed a partial stupefaction for several minutes. Impending collapse was heralded by an increase in pulse rate, a rise in the systolic and diastolic blood pressures, concentric contraction of the visual fields and failure in visual acuity of form and colour. Intense pallor of the face and marked dilatation of the pupils, which still reacted to light and accommodation, were noted. They reported a sense of alertness and stimulation after the experiment, which, associated with the rise of blood pressure, dilatation of the pupils and intense facial pallor during the exposure, suggested adrenal stimulation. The present author has experienced this syndrome with a marked euphoria after a similar exposure.

In a study by Case and Haldane (fils) (1941) (i) a subject breathed oxygen at 7 ATA (200 fsw) in the dry. After 4 minutes he suffered vertigo and malaise and reverted to air breathing. After a second exposure at 200 fsw (7 ATA) he developed twitching in the forearm and 'breathing difficulty' after 4 minutes. He returned to air breathing and decompression was commenced. Despite this he convulsed 5 minutes later. A second subject breathed oxygen for 5 minutes at 6.15 ATA (170 fsw) and developed marked restriction of vision. A third subject survived 4 minutes on oxygen at this pressure without symptoms. Four other subjects breathed

oxygen for 5 minutes at 6.15 ATA (170 fsw). One had no symptoms; one vertigo and faintness at the end, and two had 'unpleasant' respiratory symptoms at the end.

Case & Haldane (1941, (ii)) reported that about half of the subjects in these experiments stated that they could taste oxygen at 5 to 7 ATA. The taste was both acid and sweet "like dilute ginger beer." Professor Haldane, Chief Petty Officer Derrick, and the present author (unpublished data 1942), breathed oxygen for 25 to 30 seconds at 300 fsw (10 ATA). The two latter subjects could only taste what they considered to be the "rubber in the circuit." It was, in our view, the same 'taste' experienced when breathing oxygen from the circuit at 70 fsw (3.1 ATA) or at atmospheric pressure. Professor Haldane thought he could taste the oxygen at 300 fsw but was less certain than in his previous exposures. The main reason for reporting this experiment is that this is the highest pressure, known to the author, at which oxygen has been breathed by human subjects.

Behnke (1942, (i and ii)) reported that "men had repeatedly inhaled oxygen for 27 minutes at 4 ATA with no other symptoms than a sensation of "cerebral fullness" and some degree of "mental torpidity." He also reported that oxygen tolerance was greatly reduced by very moderate exercise on a bicycle ergometer (VO_2 4 x resting uptake approx.). Thus at 3 ATA average oxygen tolerance was reduced from 3 hours or more to "about twenty minutes" when subjects developed "extreme fatigue, numbness of the lower extremities and in one instance an abortive seizure." No individual exposures were described.

It will be noted that all the observations on human subjects described so far, were while breathing oxygen in compressed air.

Lorrain Smith effect: The main interest in possible pulmonary irritation and damage due to increased oxygen tensions had been in the clinical field. It was generally agreed, mainly from animal work, that tensions above 60% 1 ATA were dangerous for prolonged exposures. Fortunately, the clinical methods of administration of oxygen did not achieve consistent levels above this figure. When special efforts were made to achieve higher concentrations (near 100% 1 ATA) it was found that the intermissions on air, as recommended by Lorrain Smith, allowed such levels for several days (Boothby, Mayo and Lovelace, 1939). Becker-Freyseng and Clamann (1939) reported that one of the authors (B-F)

developed bronchopneumonia after breathing 0.9 to 1.0 atmospheres of oxygen for 60 hours. To return to our main theme, there had been no instance of pulmonary irritation in man due to hyperbaric oxygen in any form of diving (1942). Pure oxygen had not been generally used in self-contained diving and it had only been administered for short periods at relatively shallow stops (60 to 10 fsw) during decompression after air dives (Damant, 1933) or oxyhelium mixture dives (Behnke, 1942, (iii)).

Cardiovascular System: Slowing of the heart rate in the human subject at increased pressures has long been known (Bert, 1878). Benedict and Higgins (1911) reported bradycardia in young adults breathing increased percentages of oxygen. The degree of bradycardia appeared 'roughly proportional' to the percentage of oxygen breathed. Behnke et al (1935) reported that subjects breathing oxygen at 2, 3 and 4 ATA showed but little change of blood pressure except when heralding the commencement of acute oxygen poisoning when increased heart rate, rise in blood pressure and intense skin vaso-constriction occurred.

Position in Diving in 1942: The United States Navy gave times of safety when breathing oxygen as two hours at 50 fsw and thirty minutes at 90 fsw (1941). Oxygen breathing decompression stops from 60 to 10 fsw, both under water and in chambers, were being introduced at this time (Behnke, 1942 (iii)). There were no independent oxygen breathing divers. With regard to symptoms, it was stated "The first signs of oxygen toxicity are flushing of the face, nausea, dizziness and muscle twitching. A feeling of being irritable and a sense of excitement may follow." "As pressure is increased nausea, vertigo and finally unconsciousness and convulsions ensue."

The Royal Navy had used oxygen decompression from 60 fsw (submerged decompression chamber) after deep diving and from 60 fsw (deck chamber) in surface decompression for some years. There were, until 1942, no independent oxygen divers and as these were being developed, the Royal Navy followed the recommendations made by the United States Navy (see above). The fact that two Royal Naval officers had suffered acute oxygen poisoning at 90 fsw (dry) in 16 and 13 minutes appeared to have been unnoticed or forgotten. Experienced air divers were inclined to consider oxygen to be dangerous under water at depths greater than 33 fsw. This was due to the entirely mistaken view, taken in the 1930-33 Admiralty Deep Diving Trials, that the oxygen in the air (2.1 ATA at 300

fsw) caused the severe psychological disturbances encountered (see above). The instructions in the Royal Navy Submarine Escape Handbook (1942) concerning the safe times for breathing oxygen (60 min. at 100 fsw, 6 min. at 200 fsw and 3 min. at 300 fsw) appeared unrelated to any known previous investigations. With regard to the symptoms of oxygen poisoning, the Handbook stated "tingling of the fingers and toes and twitching of muscles, especially round the mouth (warning symptoms). Convulsions followed by unconsciousness and death if a remedy is not taken."

REFERENCES

BARACH, A.L. (1926): The effect of atmospheres rich in oxygen. *Am. Rev. Tuberc.*, **13**, 293-361.

BEAN, J.W. and ROTTSCHAFER, G. (1939): Reflexogenic and central structures in oxygen poisoning, *J. Physiol.*, **94**, 294-306.

BECKER-FREYSENG, H. and CLAMANN, H.H. (1939): Zur frage der Sauerstoffvergiftung., *Klin. Wschr.*, **18**, 1382-1385.

BEHNKE, A.R. (1942, (i)): Physiologic studies pertaining to deep sea diving and aviation., *Bull N.Y. Acad. Med.*, **18**, 561-585.

BEHNKE, A.R. (1942, (ii)): Effects of high pressures: prevention and treatment of compressed air illness., *Med. Clinics of N. America.*, **26**, 1213-1237.

BEHNKE, A.R. (1942, (iii)): Employment of helium to new depths of 440 feet., *Nav. med. Bull., Washington.*, **40**, 65-67.

BEHNKE, A.R., FORBES, H.S. and MOTLEY, E.P. (1936): Circulatory and visual effects of oxygen at 3 atmospheres., *Amer. J. Physiol.*, **114**, 436-442.

BEHNKE, A.R., JOHNSON, P.S., POPPEN, J.R. and MOTLEY, E.P. (1935): The effect of oxygen on man at pressures from 1 to 4 atmospheres., *Amer. J. Physiol.*, **110**, 565-572.

BEHNKE, A.R., SHAW, L. A., SHILLING, C.W., THOMSON, M.W. and MESSER, A.C. (1934): Studies in the effects of high oxygen pressure on CO_2 and O_2 content, pH & CO_2 – combining power of the blood. *Am. J. Physiol.*, **107**, 13-28.

BEHNKE, A.R., THOMSON, R.M. and MOTLEY, E.P. (1935): The psychological effects from breathing air at 4 atmospheres., *Amer. J. Physiol.*, **112**, 554-558.

BENEDICT, F.G. and HIGGINS, H.L. (1911): Effects on men at rest of breathing oxygen-rich gas mixtures., *Amer. J. Physiol.*, **28**, 1-28.

BERT, P. (1878): *La Pression Barometrique, G. Masson, Paris.*

BOHR, D.F. and BEAN, J.W. (1940): Dehydrogenase inactivation in oxygen poisoning, *Amer. J. Physiol.*, **131**, 388-393.

BOOTHBY, W.M., MAYO, C.W. and LOVELACE, W.R. (1939): Oxygen and oxygen and helium therapy: recent advances., *Med. Clin. N. Amer.*, **23**, 977-1005.

BORNSTEIN, A and STROINK, M. (1912): Ueber Sauerstoff vergiftung. *Dtsch. med.* Wschr., **38**, 1495-1497.

BORNSTEIN, A. (1910): Versuche uber die Prophylaxe der Pressluftkrankheit. *Pflug, Arch. Geo. Phys.*, **4**, 1272-1300.

CAMPBELL, J.A. (1929): Effects of breathing oxygen at high pressures, *J. Physiol.*, **68**, 7-8.

CASE, E.M. and HALDANE, J.B.S. (1941, (i)): Human physiology under high pressure., *J. Hyg. Camb.*, **41**, 225-249.

CASE, E.M. and HALDANE, J.B.S. (1941, (ii)): Tastes of oxygen and nitrogen at high pressures., *Nature*, **148**, 84.

DAMANT, G.C.C. (1930): Physiological effects of work in compressed air. *Nature, London.*, **126(2)**, 606-608.

DICKENS, F. (1945): *Personal communication, since published.*

DICKENS, F. (1946): Toxic effects of oxygen on brain metabolism and tissue enzymes, *Biochem. J.*, **40**, 145-187

FAULKNER, J.M. and BINGER, C.A.L. (1927): Oxygen poisoning in cold blooded animals, *J. Expl. Med.*, **45**, 865-872.

GERSH, I. (1944): Oxygen poisoning, *U.S. Navy Research Project x 192.*

HALDANE, J.S. and PRIESTLEY, J.G. (1935): *Respiration, Oxford.*

HILL, L and MACLEOD, J.P. (1903): The influence of compressed air on the respiratory exchange, *J. Physiol.*, **29**, 492-510.

HILL, L. and PHILLIPS, A.E. (1932): Deep Sea Diving, *J. R. Nav. Med. Serv.*, **18**, 155-173.

LAVOISIER, A.L. and SEGUIN, A. (1789): Premier memoire sur la respiration des animaux. *Hist. Acad. Sci., Paris*, **185**, 566-584

LIBBRECHT, W. and MASSART, L. (1937): Influence de l'oxygène sous pression sur la succino dehydrogenase. *Compt. rend. Soc. de Biol.*, **124**, 299-300.

MASSART, L. (1936): L'oxydose et le cytochrome, *Arch. int. Pharmacodyn.*, **53**, 562-568.

QUASTEL, J. H. (1939): Oxygen poisoning, *Physiol. Rev.*, **19**, 135-145.

REGNAULT, V. and REISET, J. (1849): Recherches chimiques sur la respiration des animaux de diverses. classes. *Annals de Chimie et de Phys*, **26**, 299-519.

REPORT of a Committee appointed by the Lords Commissioners of the Admiralty to consider and report upon the conditions of Deep Water Diving, Lond, 1933.

SHILLING, C.W. and ADAMS, B.H. (1933): A study of convulsive seizures caused by breathing oxygen at high pressures, *U.S. Naval Med. Bull., Washington*, **31**, 112-121.

SMITH, J. LORRAIN (1899): The pathological effects due to increase of oxygen tension in air breathed, *J. Physiol.*, **24**, 19-35.

STADIE, W.G., RIGGS, B.C. and HAUGAARD, N. (1944): Oxygen poisoning, *Am. J. of Med. Science*, **207**, 84-114.

THOMSON, W.A.R. (1935): The Physiology of Deep Sea Diving. *British Medical Journal.*, **2**, 208-210.

CHAPTER 2

OXYGEN POISONING STUDIES 1942-5

In 1942 independent oxygen diving for various covert operations was introduced in the Royal Navy on a large scale and a number of incidents had occurred (see Appendix 1). The generally accepted times of safety when breathing oxygen were based on remarkably few experiments in which oxygen was breathed, usually at rest, in compressed air. No controlled experimental dives where men had breathed pure oxygen at different depths under water had ever been carried out.

The objects of the experiments described here was to gain a more comprehensive and accurate picture of the reaction of the human subject to toxic tensions of oxygen particularly under water. Large groups of subjects were therefore employed and over two thousand experiments carried out. In many experiments the subjects were wearing diving suits and were under water to determine whether the present accepted times and rules of safety gave an accurate assessment of the dangers of high tensions of oxygen when diving in open water.

The Marked Variation of Oxygen Tolerance in Man

The first series of experiments to be carried out was to determine the oxygen tolerance of a group of healthy subjects at a fixed oxygen tension (90 fsw, 3.71 ATA).

Method: This series was carried out in a dry pressure chamber of 100 cubic feet capacity. The subject was seated with his knees slightly flexed and his back rested against the side of the tank. He was well illuminated and had two trained observers seated opposite to him. The attendants were in constant telephonic communication with those outside. The subject continued to breathe oxygen until marked symptoms occurred.

Experiments in compressed air have a number of advantages. The subject's condition can be observed carefully. This is impossible if the individual is in a diving suit underwater. He gains confidence from the immediate proximity of the observers and can refer any doubts as to his condition by means of pointing or by notes to the attendants. Subjective end-points are less likely. If a sudden or violent end-point occurs, then oxygen breathing can be immediately discontinued either by the subject or the attendant removing the mouthpiece. Convulsions are a lesser risk in a well cushioned dry compartment with two attendants.

Oxygen was breathed from a 'Salvus' apparatus. This has a simple counterlung, a one pound canister, and employs pendulum breathing. Oxygen is supplied automatically by a reducing valve. The rate of flow in these experiments was 0.75 litres per minute. This was in excess of the amount required (0.3 l/min. approx.), and the washing out of the apparatus was thus assured. Excess pressure in the counterlung could be relieved by a manually operated release valve. A mouthpiece and nose clip were used in preference to a mask as there is less risk of air leaking into the circuit and the subject's face is less obscured. The apparatus was carefully checked and the absorbent canister filled with fresh soda-lime immediately before each experiment. The oxygen employed throughout these experiments was supplied by the British Oxygen Company, and is guaranteed 99.8 to 99.9% pure, the residue being inert gases. The oxygen inhaled never had a percentage of carbon dioxide exceeding 0.1%. The subject did not breathe oxygen until the chosen depth (90 ft. of sea water, 3.72 atmospheres absolute) was reached, by allowing air to run from a bank of high pressure air cylinders into the chamber.

The rinsing out of nitrogen from the lungs was carried out by inhaling oxygen deeply from the apparatus and exhaling through the nose. This was carried out six times, note being made that the counterlung was sucked empty. Washing out was repeated every five minutes for the first half hour, and every ten minutes after that time. This was in order to eliminate any nitrogen rinsed out from solution in the tissues in the body or diffusing or leaking into the apparatus. In most exposures the pulse and respiration were noted every five minutes.

A series of analyses showed that, even with frequent washing out, the percentage of oxygen breathed in the dry varied between 92 and 97%.

Subjects varied from recently trained divers to experienced divers,

submarine ratings, medical officers, special service operational personnel and mine disposal officers and ratings. All these officers and ratings were grade A1 in fitness. Ages varied from 18 to 40 years.

The attendants breathed air and were decompressed according to Boycott, Damant and Haldane's air tables. In an emergency the whole party was immediately surfaced and the subject was removed for medical attention. As he had been breathing oxygen, it was considered that there was no risk of bubble illness. The attendants were rapidly recompressed before bubble formation could occur.

Experiments carried out in this manner are referred to hereafter as 'in the dry' in contrast to those carried out under water and referred to as 'in the wet.'

Results: These are given in Table 1 (Appendix 2) and illustrated in Figure 1. Times on oxygen at 90 fsw (3.72 ATA) are shown before the subjects developed symptoms necessitating the cessation of the exposure. Five subjects convulsed the rest recovered on being turned on to air. The nature and time of occurrence of earlier symptoms are also given in Table 1.

The most striking finding was the enormous variation in oxygen tolerance in a group of human beings. Exposures, causing marked symptoms at this tension, varied from 6 to 96 minutes in a group of 37 individuals. The tolerance of each subject was unpredictable. It will be noted that the age, weight and height of all subjects is given (Table 1). Many attempts to correlate these various data with oxygen tolerance in a group of 26 have not been successful. Physical fitness, athleticism, smoking, ingestion of alcohol, and psychological health, did not appear to influence this tolerance.

At this depth the interval between early and 'terminal' symptoms varied from 0 to 55 minutes. It is possible that some of the earlier fibrillary tremors of the lips were due to nervousness or to the strain of holding the mouthpiece. Symptoms will be discussed in detail in a later section. There is strong evidence that there is an individual variation, not only in the time of exposure tolerated, but in the particular portion of the central nervous system first to be so affected that it shows overt signs of dysfunction.

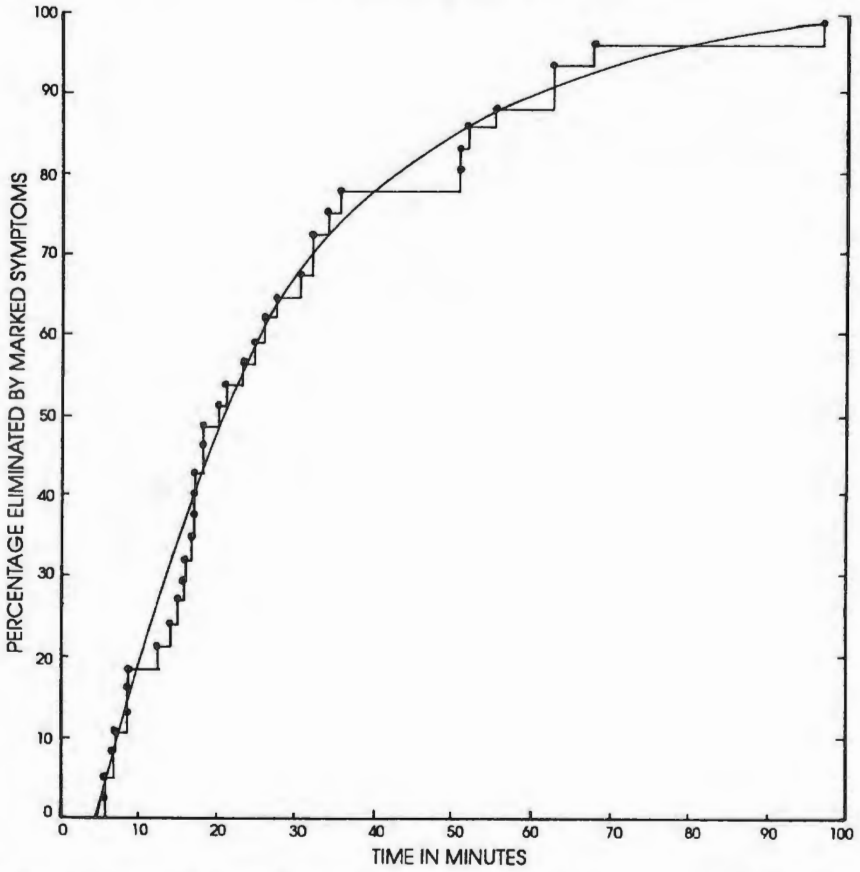

FIG. 1: Percentage of subjects in a group of 37 eliminated by toxic symptoms, as a function of the time of exposure to pure oxygen at 90 feet in compressed air. No work performed. Actual end points plotted.

In Fig. 1, the percentage of subjects eliminated by symptoms is plotted against time of exposure. This type of skew distribution was first described by Galton and Macalister. Notable examples are the response of animals to drugs and hormones, and the susceptibilities of insects to poisons (Bliss). In such distributions the logarithms of the dosages, needed to produce a given effect (e.g. to cause oestrus or death), are normally distributed. Small experimental animals i.e. rats when studied in groups, appear to have a greater resistance to oxygen poisoning than man, but it must be remembered that in animal experiments convulsions are avoided if possible. Many animals show signs of poisoning, such as twitching and

abnormal respiration, for some time at similar pressures before actually passing into convulsions. The greater resistance of experimental animals may be illusory.

It is clear that the previously reported times of safety at this depth are dangerously incorrect. No allowance whatsoever, had been made for individual variation which is found to be over an enormous range.

Oxygen Tolerance in Man Under Water at 50 fsw (2.5 ATA)

It has already been emphasised that, up to the commencement of these investigations, all experiments regarding tolerance to oxygen under pressure had been carried out by subjects in dry chambers. This series of dives was initiated to discover whether man's tolerance was similar, under water, to that so far determined in compressed air.

Method: The diver in these experiments wore a light rubberised canvas suit with a soft helmet, (see Fig.2). The respiratory apparatus was a considerable modification of the well known Davis Submarine Escape Apparatus. It was adapted for very much longer performances by the insertion of a 4 lb. radial canister, containing carbon dioxide absorbent and by the attachment of two 2-litre bottles of oxygen, charged to 120 atmospheres (over four hours endurance). Oxygen was supplied by means of a reducing valve at the rate of 1.2 litres per minute. The excess of oxygen being supplied over the actual consumption of the resting diver (0.25 to 0.35 1/min. approx) assured the constant rinsing out of his lungs and respiratory apparatus. As the counterlung had an automatic non-return exhaust valve, no discomfort was caused to the diver.

The diver carried out the usual washing out process (see previous section) to ensure that his lungs and apparatus were filled with oxygen. He was then submerged in an open tank and tested for leaks. When his suit and apparatus were considered satisfactory he walked to the high pressure tank and was lowered into the water. The upper hatch was closed and bolted and pressure rapidly applied. On the average, subjects were breathing oxygen for ten minutes before they arrived at the appropriate experimental depth, about eight minutes being at, or near, atmospheric pressure.

"Human Torpedo" oxygen diver. Note soft helmet and vizor. Diver has mouthpiece and nose clip. Pendulum breathing into counterlung through large radial carbon dioxide absorbent canister. Luftwafe duraluminium oxygen cylinders carried on back.

The pressure tank employed in these experiments was 12 ft. in height and 6 ft. in diameter (see Fig. 2). It was filled with water to a depth of 8 ft. The diver was lowered through the upper hatch into the water. Depth was simulated by increasing the air pressure above the water. An attendant sat on a ledge inside the chamber breathing air and was in telephonic communication with the outside attendant. He wore waders and sat above a ladder running down into the water in which his legs were immersed. He held a lifeline running to the diver. The temperature of the water was maintained at 65°F. The depth reading in these experiments was actually of the air pressure above the water. Thus the pressure at which the gas was being breathed was approximately 2 to 3 ft. of water greater than the gauge reading. In open water, on the other hand, depth is estimated by lead or echo sounding, and, under these conditions, the operational depth (booted diver) would be 8 ft. greater than that measured in the pressure tank. (see Fig. 2)

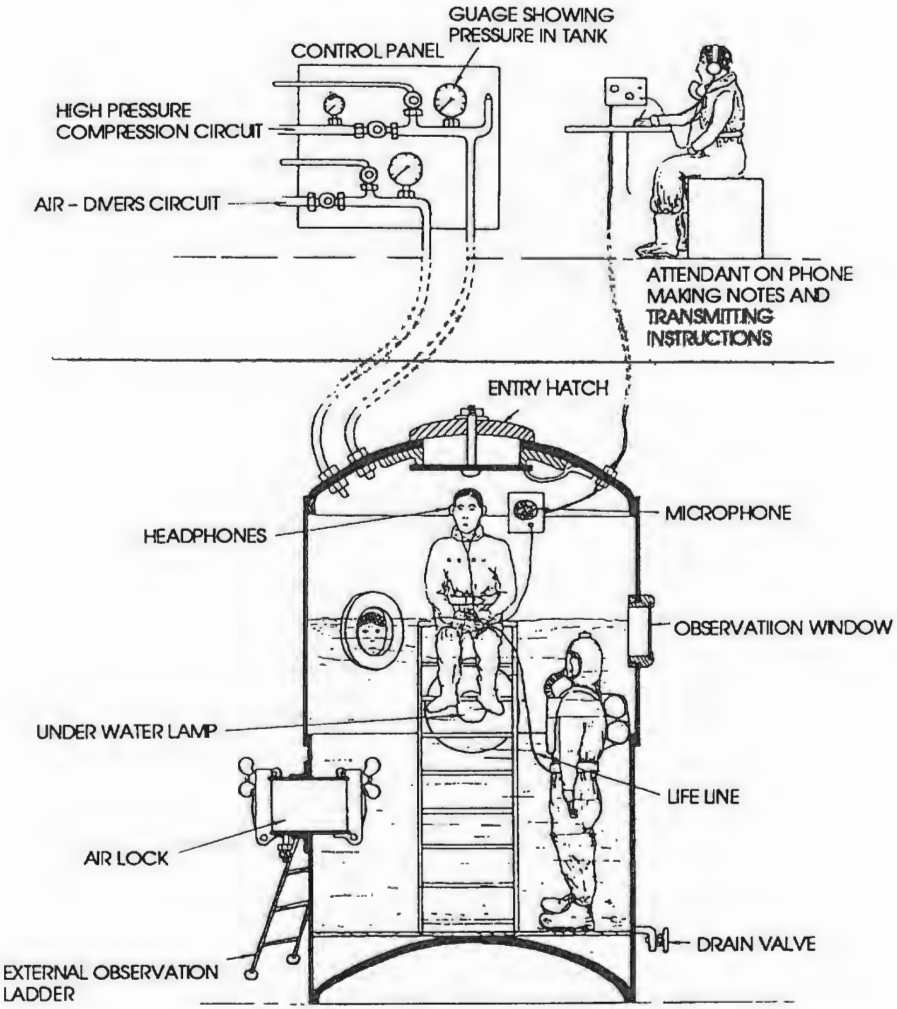

FIG. 2 **Showing wet pressure chamber with diver under water breathing oxygen in self-contained set. Internal and external attendants.**

The first series of dives was to 50 ft. of sea water (2.5 atmospheres absolute) and the time limit was 30 minutes. 100 dives were carried out. The large majority of the divers were special service operational personnel and with a few exceptions, the divers completed their half hour or had convulsive symptoms. If the diver convulsed or had severe symptoms he was hauled up by the attendant and turned on to air. The pressure was immediately released.

Welcome to the wet pot. Attendant above water breathing oxygen-nitrogen mixture to allow immediate decompression if necessary.

Attendant in wet pot instructing human torpedo oxygen diver just before going underwater.

Wet Chamber sealed. Outside attendant gets "OK" signal. Pressurization starting.

Oxygen diver being hauled out of wet pot. Vizor open and turned onto air. Airway being checked.

The mouthpiece acted as an excellent gag during convulsions and attendants were taught to hold the head correctly to maintain a good airway. The morale of these men was obviously of a very high order (see Appendix 1). Results are shown in Table 2 (see Appendix 2). Out of 100 divers, 26 convulsed, another 24 had symptoms and 50 had no symptoms.

It will be noted that the degree of exercise was not carefully controlled and mention is made in each dive as to whether any exercise was undertaken during the exposure. The degree of exercise was variable and, in some cases, for a short period or intermittently.

If we divide the dives into those exercising and those not exercising, we obtain the following:

Divers Exercising (n = 37)

8 (22%) convulsed in 10 to 30 minutes
8 (22%) lip twitching in 9 to 30 minutes
2 (5%) malaise, nausea, respiratory distress, in 11 to 31 minutes
19 (51%) no symptoms during 30 minute exposure.

Divers Not Exercising (n = 63)

18 (28%) convulsed in 7 to 29 minutes
13 (21%) lip twitching in 10 to 30 minutes
32 (51%) no symptoms during 30 minute exposure

These findings did not suggest that exercise increases oxygen toxicity but this aspect is studied more specifically later in this study.

These divers were operational personnel and not experimental divers. They considered, commendably, although quite incorrectly, that oxygen at toxic pressure was another trial to be undergone before selection was completed. Symptoms are therefore minimal.

According to previously accepted figures, men were safe breathing oxygen at this depth for two hours. It is obvious that the variation of tolerance, demonstrated in the previous section, made these figures unreliable, as they were inferred from a few experiments in the dry. The same variability is shown in this series although an exposure of 30 minutes only gave symptoms in 50% of the group. However, the total result, even allowing for variation, strongly suggests a marked decrease of tolerance

under water to that obtaining in the dry, whether the diver is resting or exercising.

The Oxygen Tolerance of Man at Rest in Compressed Air and Under Water

A series of resting dives was carried out to compare the tolerance of subjects to oxygen at increased tensions in compressed air and under water. The experiments were performed under conditions similar to those already described at both 60 and 90 fsw, (2.82 & 3.73 ATA respectively). Dives were to end-point but exposures in the dry were limited to two hours unless the subject volunteered to continue.

The following results were obtained. The time of exposure in minutes and symptoms are given (see also Fig. 3).

COMPARISON OF OXYGEN TOLERANCE IN THE WET AND IN THE DRY, AT 60 FEET AND 90 FEET, SEA WATER

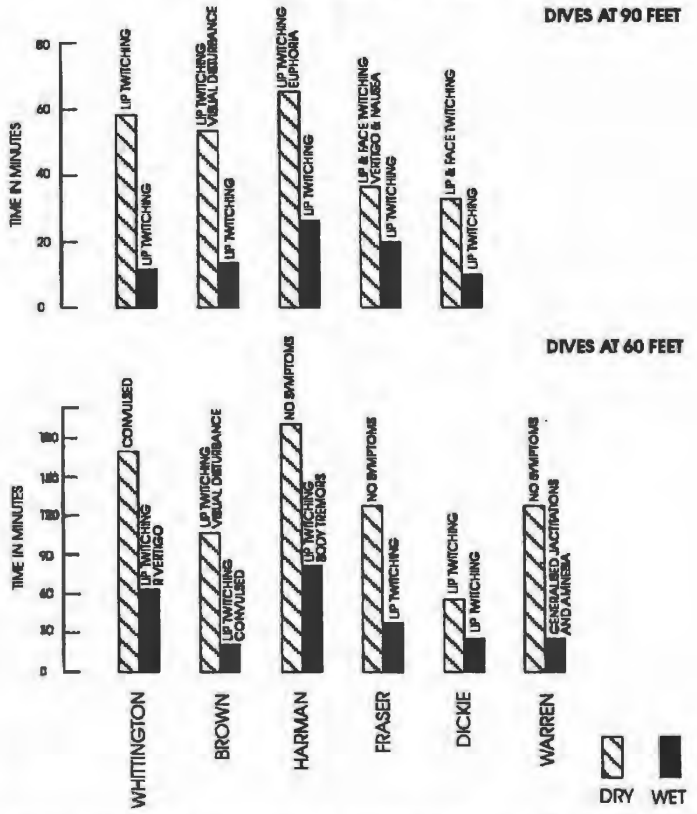

FIG. 3: Showing the marked impairment of oxygen tolerance in individuals in the wet as compareed with in the dry. Dives at 60 feet and 90 feet with end points are shown.

TABLE 3

60 FEET OF SEA WATER (2.82 ATA)

	DRY	WET
Warren	120, felt heavy and dull during last hour	25, lip twitching at end, looked 'out of touch'. Generalised jactitations for 20 seconds after being turned on to air. Retrograde amnesia.
Whittington	158 convulsed. No warning.	61, vertigo at 36 minutes, slight lip twitching at 55 minutes. Severe lip twitching at end.
Brown	101, extreme sleepiness at 20 minutes. Severe lip and nose twitching and marked 'diaphragmatic spasm' at end.	19, lip twitching at end. Convulsed 30 seconds after being turned on to air.
Harman	180, no symptoms	76, violent 'shivering' last 5 minutes. Lip twitching at end.
Fraser	120, twitching of upper lip at 100 minutes. No other symptoms except transient vertigo on reverting to air	37, lip twitching at end.
Dickie	51, slight lip twitching at 33 and 50 minutes. Sudden severe lip twitching at end	12, severe lip twitching last two minutes of exposure

90 FEET OF SEA WATER (3.73 ATA)

	DRY	WET
Whittington	54, sustained lip twitching last 4 minutes. Very severe at end	11, severe lip twitching at end
Brown	51, slight lip twitching at 43 minutes. Increasingly severe lip twitching last 8 minutes. Visual symptoms lateral movement of images and bright dazzling flashes	12, sudden severe lip twitching at end
Harman	62, pouting of upper lip 2 minutes before end. 'Pleasant sensation of intoxication' with marked euphoria	11, severe lip twitching at end
Fraser	34 fine lip twitching after 27 minutes. Convulsive movements of whole face at end. Slight nausea and vertigo during last 6 minutes	18, lip twitching at end
Dickie	32, lip twitching after 29 minutes which spread to whole of lower face and increased in severity	9, severe lip twitching last 20 seconds

Dry/Wet Ratio

It is of interest to examine the ratios of times of tolerance in the wet and in the dry.

	Series at 90 fsw	Series at 60 fsw
Brown	4.25	5.3
Whittington	5	2.6
Harman	2.4	2.4 plus
Dickie	3.4	4.1
Fraser	1.9	3.2
Warren	–	4.8 plus

It thus appears that, when oxygen is breathed under water, the period of time before toxic symptoms occur is markedly less than that which would obtain breathing oxygen in compressed air at the same pressure. This is further demonstrated with large groups later in this presentation.

The enormous importance of this finding in relation to free oxygen diving need hardly be pointed out. The degree of impairment of tolerance in the wet as compared with that in the dry is certainly far from being fully explained by the slight dilution by nitrogen of the oxygen breathed in the dry experiments (DO_2 of the order of 56 fsw in 60 fsw exposures and

of 84 fsw in the 90 fsw exposures). In the Royal Navy oxygen has only been breathed in compressed air during decompression from deep air dives (1932) when the diver enters a submerged decompression chamber and breathes oxygen to accelerate nitrogen elimination from his body. The greatest depth at which oxygen is thus breathed is at 60 fsw in the dry and then only for short periods. Thus the new use of oxygen for free covert operational diving in the Royal Navy (1942) requires urgent definition of the unexpected dangers of breathing oxygen under water.

The causes of the decreased tolerance under water remain unknown. Carbon dioxide accumulation was suspected but numerous analyses of the gas in the counterlung, of the inspired and alveolar gases negated this possibility. The highest carbon dioxide content of the inspired gas found was 0.2% (one atmosphere) and in the large majority of cases was far less. The lack of a rigid helmet, respiratory resistance, the bandaging effect of the suit, the diver's posture and hydrostatic effects in general have all been investigated to a greater or lesser extent with negative results. These experiments are not described here as they are incomplete.

Time/Pressure Relationship for Men Breathing Oxygen Underwater, Resting

The next series of experiments was an attempt to obtain a series of curves for various individuals, giving the relation of the depth of the diver in water to the time of survival when breathing pure oxygen. In view of the variation between individuals, it was realised that each of the five divers would have a different curve of safety at various depths.

Method: This series was carried out in the wet pressure chamber as described above. Each subject was dived to 50 ft., 60 ft., 70 ft., 80 ft., 90 ft. and 100 ft. In the case of the more oxygen sensitive subjects, a few more shallow dives were included. Divers were instructed to surface and to report to the attendant immediately they felt severe lip twitching or any symptoms that justified the termination of the exposure. The attendant turned the diver on to air if he considered that he was in danger of convulsing. Divers were always rested the next day and rarely carried out more than two dives per week. If a diver convulsed he was given a "stand off" for several days.

Results: Table 4 shows the results obtained (see Appendix 2). These are plotted in Figs. 4 to 8. The dives were over a period of 50 days and the

number of the day in this period is shown against each dive. The asymptote of the curve for each diver obviously depends on the maximum non-toxic depth of that particular diver. This is difficult to demonstrate as conditions in the wet pressure tank are not altogether pleasant and two or three hours endurance is about as much as one can reasonably expect.

At this stage of the investigation a new factor was becoming increasingly manifest. It was apparent that the tolerance of an individual diver varied from day to day. Thus, if an attempt is made to plot the time of tolerance against the depth, a third factor enters, that is the variation of tolerance of the diver over the period of experiments. The small inset graph in Figs. 4 to 8 is a very rough hypothetical representation of the behaviour of each subject's tolerance over this period.

Brown: (Fig.4). This diver gives an 'excellent' curve, extremely reminiscent of that obtained by Hederer and André (1940) with rabbits. The most 'aberrant' point is the dive to 80 ft. This was repeated (square symbols for repeats).

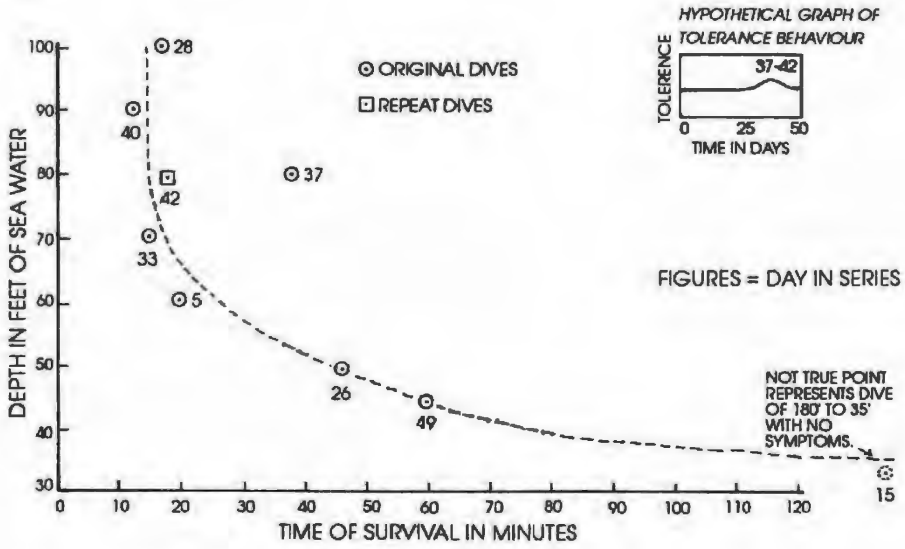

FIG.4: BROWN

Harman: (Fig. 5). This diver showed a constant high level of tolerance. There is a suggestion that his tolerance was steadily improving throughout the period.

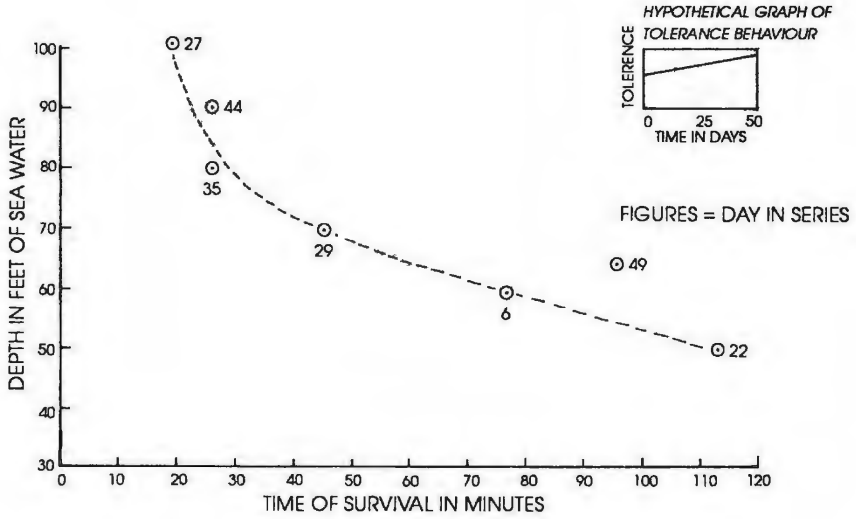

FIG. 5: HARMAN

Dickie: (Fig. 6). This series of dives, when plotted, shows marked 'scatter' although his general tolerance was high.

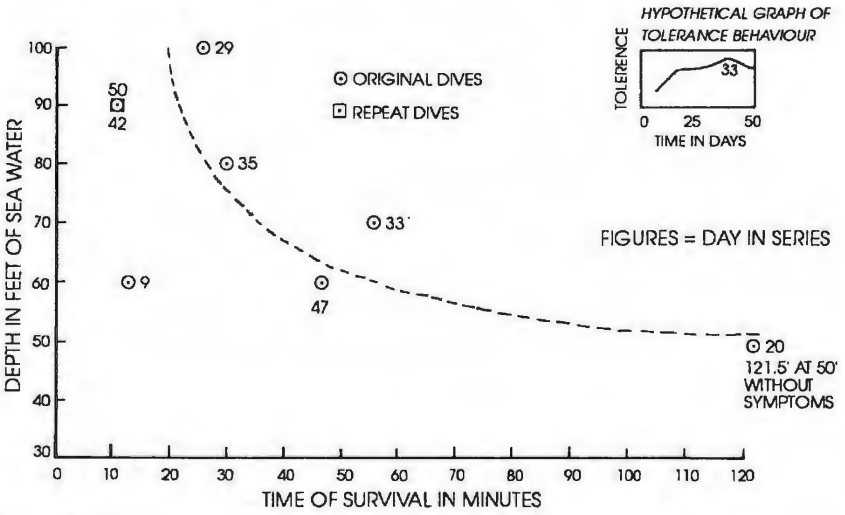

FIG. 6: DICKIE

Fraser: (Fig. 7). This diver gave a 'good' curve, with fairly sustained resistance to the toxic effect of oxygen. However, the time of survival at 60 ft. on the seventh day was markedly below the rest of his performances. A

repeat dive to this depth and another dive to 70 ft. showed that his tolerance had fallen away again.

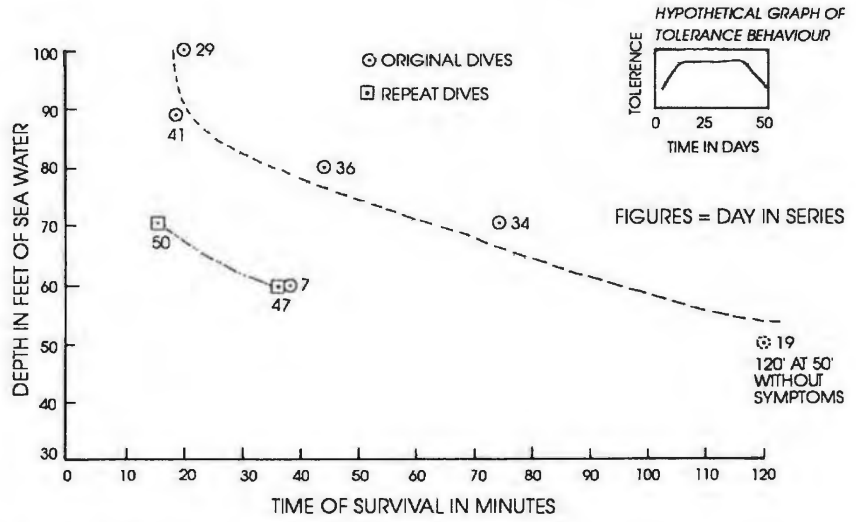

FIG. 7: FRASER

Whittington: (Fig.8). This diver showed a reasonably 'good' curve up till about the 37th day, when his tolerance appeared to fall markedly. Repeated dives at 60 ft. and 50 ft. showed that this subject had gone into a new and very inferior curve.

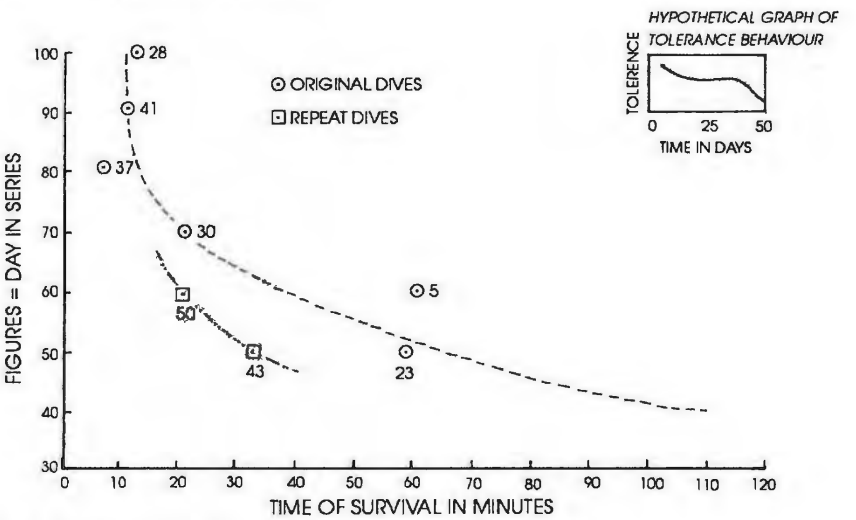

FIG. 8: WHITTINGTON

It is apparent that an attempt to plot a curve giving times of safety when breathing oxygen at various depths, for even a single diver, is quite impossible owing to his variation of tolerance from day to day. It appears that certain divers show this individual variation to a greater degree than others. As with the variation between individuals, no cause could be found of this varying susceptibility to oxygen at toxic tensions. The divers, in the series described above, were in excellent health throughout. Careful enquiries into their habits, amount of sleep, smoking, ingestion of alcohol, diet, times of meals, etc., elicited no significant factor.

Figure 9 shows a mathematical curve (time/depth) that fits approximately the medium values for each depth. It is a rectangular hyperbole representing the relation:

$$T = \frac{1{,}086}{d - 39.7}$$

where T = time of survival in minutes

d = depth of water in feet

MATHEMATICAL CURVE – APPROXIMATING ROUGHLY TO THE MEDIAN VALUES OF SUBJECTS IN FIGS 4 TO 8

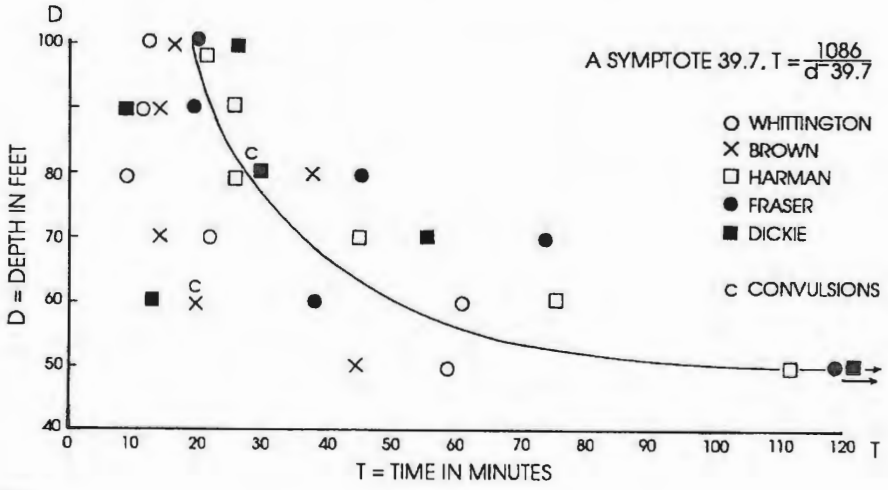

FIG. 9

The Variation of Oxygen Tolerance of the Individual

In view of the findings of the previous section, a series of under water dives were carried out to determine the degree of variation of tolerance of an individual diver breathing oxygen at a constant depth on different occasions.

Method: A diver of apparently good resistance to oxygen poisoning was selected. He dived twice a week, over a period of three months, to a constant depth of 70 ft. in the wet (65°F). On all occasions he wore the same light diving suit and self-contained oxygen breathing apparatus. No exercise was carried out by the diver who stood on bottom in very slight negative buoyancy. All dives were carried out about 11 am. after an early breakfast. His end-points were usually very definite and his health excellent throughout.

Results: These are shown in Table 5 and Figs. 10 and 11. They show that his tolerance over the period varied within a very large range.

TABLE 5

TOLERANCE OF A SINGLE DIVER AT 70 FT. IN THE WET OVER A PERIOD OF 90 DAYS

DAY IN SERIES	TIME (MINS)	SYMPTOMS
1	7	Slight lip twitching, becoming more severe
7	12	Nausea
9	86	Auditory hallucination of loud banging and lip twitching
15	27	Increasingly severe lip twitching
17	23	Slight lip twitching, severe at end
30	28	Severe lip twitching
34	61	Intermittent lip twitching, increasing in severity
37	148	Feeling "crosseyed", lip twitching
42	37	Lip twitching, coughing
44	96	Lip twitching, stertorous breathing
48	31	Severe lip twitching
56	67	Lip twitching

DAY IN SERIES	TIME (MINS)	SYMPTOMS
70	62	Slight lip twitching for 20 minutes. Tinnitus, palatal and pharyngeal spasm, spasmodic respiration, confusion
72	43	Severe lip twitching
76	41	Lip twitching, vertigo, dazzle
78	82	Lip twitching, dazzle, dyspnoea
80	29	Lip twitching, nausea
83	125	Dazzle, amnesia
90	78	Nausea, severe lip twitching

VARIABILITY OF OXYGEN TOLERANCE OF A SINGLE DIVER

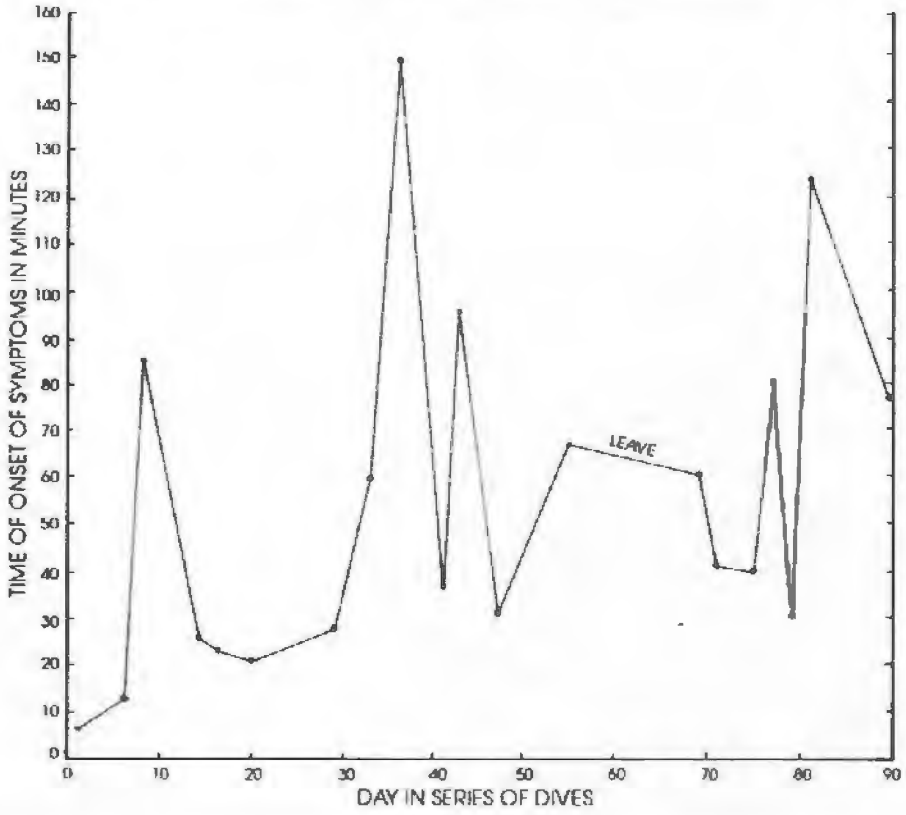

FIG. 10: Time of exposure causing toxic symptoms in the same diver under water at 70 feet of sea water over a period of 90 days. Temperature of water 65°F. No work performed.

VARIATION OF OXYGEN TOLERANCE OF SINGLE DIVER

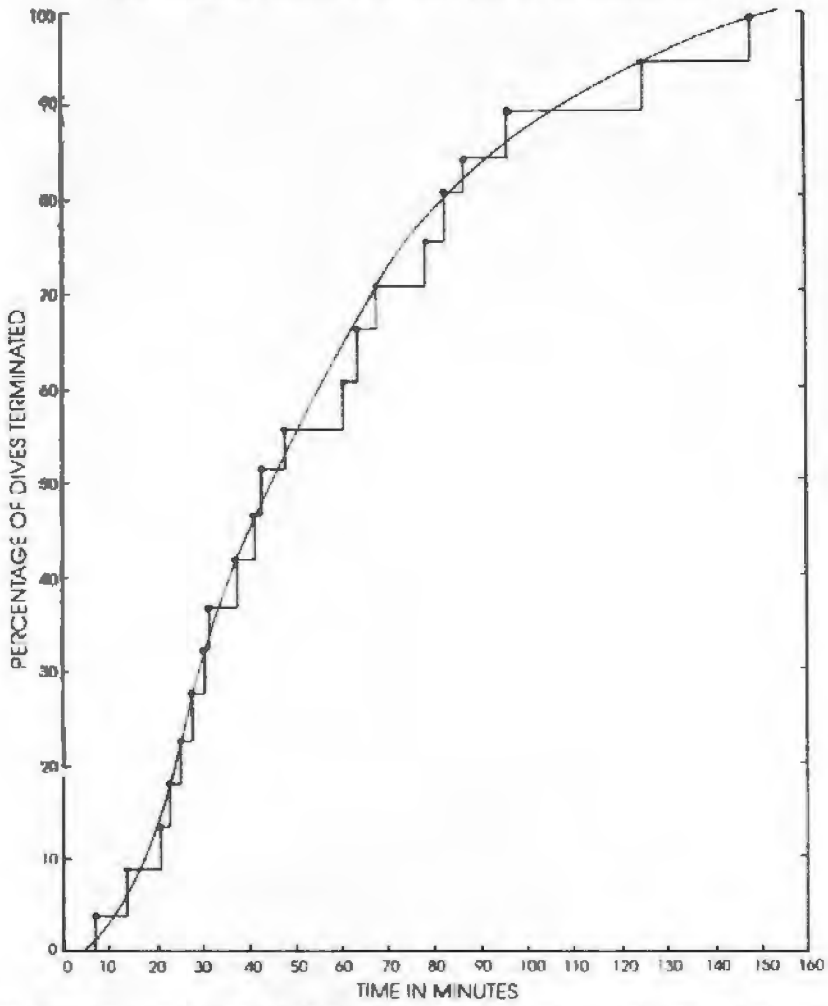

FIG. 11: Percentage of dives terminated owing to toxic symptoms as a function of duration of exposure. Temperature of water 65°F. No work performed. Depth throughout 70 feet of sea water. Dives over a period of 90 days.

Fig. 11 shows his dives plotted in order of performance as if they were carried out by twenty different divers. A curve was obtained very similar to that showing variation in the tolerance of a group of individuals. It appears from statistical analysis, however, that the diver selected for this series showed a greater variation of tolerance than the average.

A further series of repeated dives were carried out to 50 ft. by eight subjects and to 70 ft. by two subjects. Their performances are tabulated below:

TABLE 6

VARIATION OF TOLERANCE OF INDIVIDUAL DIVERS AT 50 FT. AND 70 FT. UNDERWATER, REST.

NAME	TIME (mins)	DEPTH (fsw)	SYMPTOMS	DAY IN SERIES
Gibson	12	50	Convulsed	1
	100	50	No symptoms	17
	23	50	Convulsed	23
	10	50	Lip twitching	28
		Av. 36		
		Varn. 10-100		
Gray	10	50	Severe lip twitching	1
	29	50	Slight lip twitching, body tremors	7
	25	50	Severe lip twitching	14
	21	50	Lip twitching	18
	28	50	Lip twitching, convulsed	21
		Av. 26		
		Varn. 10-29		
Knight	17	50	Lip twitching, body tremors	1
	32	50	Nausea and lip twitching	5
	24	50	Lip twitching	15
	21	50	Nausea, lip twitching	20
	39	50	Severe lip twitching	22
		Av. 27		
		Varn. 17-39		
McInnes	19	50	Lip twitching	1
	100	50	No symptoms	6
	29	50	Lip twitching	7
	20	50	Lip twitching	13
	19	50	Lip twitching	14
		Av. 38		
		Varn. 19-100		
McLaughlin	24	50	Lip twitching, convulsed	1
	53	50	Lip twitching	9
	16	50	Lip twitching	17
	27	50	Lip twitching	21
		Av. 30		
		Varn. 16-53		
Murton	102	50	Severe nausea	1
	20	50	Headache	5
	14	50	Lip twitching	10
	22	50	Lip twitching	12
		Av. 37		
		Varn. 14-102		
Shields	51	50	Severe lip twitching	1
	69	50	Convulsed	3
	20	50	Lip twitching	8
	24	50	Lip twitching	10
		Av. 41		
		Varn. 20-69		
Witham	15	50	Convulsed	1
	26	50	Lip twitching	21
	16	50	Lip twitching	24
	14	50	Lip twitching	29
	23	50	Lip twitching	
		Av. 19		
		Varn. 14-26		
		70		

NAME	TIME (mins)	DEPTH (fsw)	SYMPTOMS	DAY IN SERIES
Miller	36	70	Lip twitching	1
	68	70	Lip twitching	4
	51	70	Lip twitching	7
	44	70	Nausea	13
	43	70	Retrosternal pain and malaise	15
	32	70	Convulsed	28
		Av. 49		
		Varn. 32-68		
Herrett	36	70	Lip twitching	1
	52	70	Lip twitching	6
	26	70	Lip twitching	8
	20	70	Convulsed	12
	18	70	Lip twitching	14
	7	70	Lip twitching	19
	36	70	Nausea	27
	16	70	Paraesthesia	33
		Av. 26		
		Varn. 7-52		

It becomes clear that to judge even a single man's tolerance by one or even several dives is dangerous and unjustifiable. If we examine the performances of the three divers who survived for 100 minutes at 50 ft., we find that the averages of all their other performances at this depth are 22, 19, and 15 minutes respectively. One of the most striking cases is that of Gibson who convulsed after 12 minutes at 50 ft. 16 days later he completed 100 minutes without symptoms. 6 days after this he again convulsed at 50 ft. after 32 minutes. Such findings as this make it clear that to dive on oxygen to any toxic pressure involves a risk that is impossible to assess.

The Oxygen Tolerance of Groups of Men at Various Depths Under Water at Rest

It is clear from the above experiments that it is extremely dangerous to give any fixed times of safety for an oxygen diver at any particular depth. The variation between individuals and the variation of tolerance of each individual, make any generalisation impossible.

In this series of experiments, dives on oxygen were carried out in the wet to a definite end-point, by groups of subjects at: 50, 60, 70, 80, 90 and 100 feet in an attempt to obtain a clearer overall picture. No work or exercise was carried out during these dives.

SURVIVAL OF DIVERS ON PURE OXYGEN AT VARIOUS DEPTHS UNDER WATER

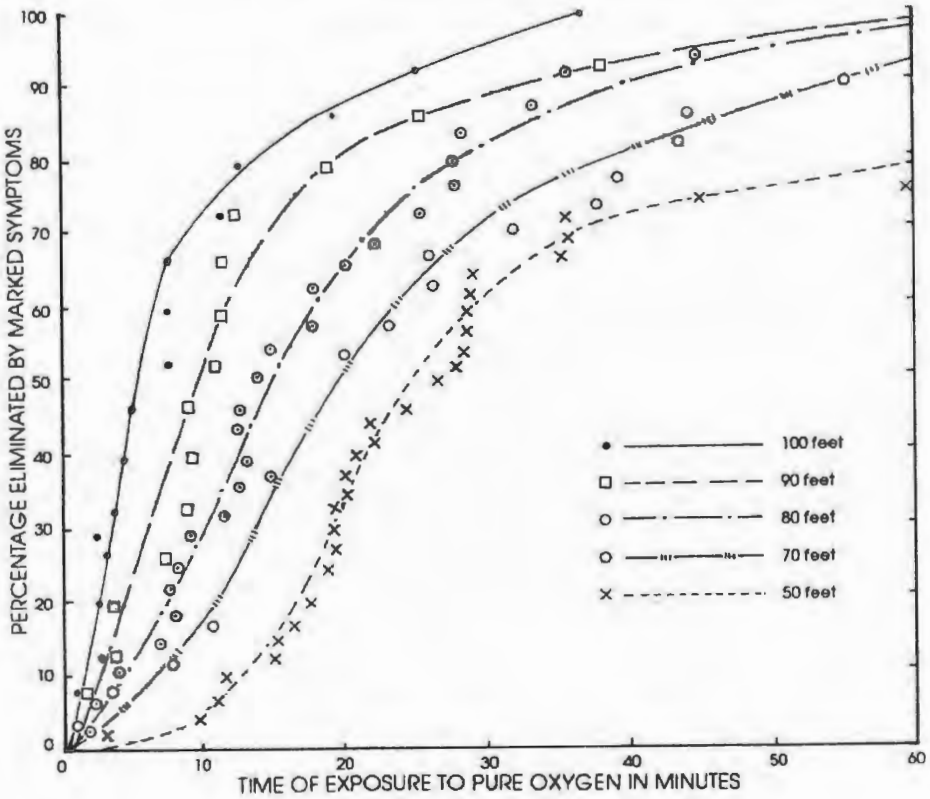

FIG. 12: Showing the percentage of divers on oxygen under water surviving at various times up to 1 hour at 50 feet, 70 feet, 80 feet, 90 feet and 100 feet of sea water. Temperature throughout 65°F. No work performed actual end points plotted.

Results: Results obtained are given in tables 7-12 (Appendix 2). In Fig. 12 the percentage of the group eliminated by severe symptoms, or convulsions, is plotted against the time of exposure at each depth. The increased toxicity of oxygen, as the depth becomes greater, is clearly shown. The highly skew distributions conform satisfactorily to the Galton – Macalister law, and it can be demonstrated that all the curves in Fig. 12 are the same curve except for a change of scale depth. In other words, variability of the group is independent of the depth. The upper parts of the curves are somewhat 'erratic' as they depend upon single performances of one or two highly resistant individuals.

Coefficient of Variation: The following table (13) shows the average and coefficient of variation of the tolerance times, in minutes, at different depths before marked symptoms arose. The series of dives to 90 ft. in the dry are included. The enormous coefficient of variation (75 to 108%) will be noted.

TABLE 13

NUMBER OF SUBJECTS	DEPTH IN FEET	AVERAGE	COEFFICIENT OF VARIATION %
40	50	43.2	104.4
18	60	29.5	81.2
24	70	26.8	75.8
27	80	19.1	80.1
15	90	16.3	108.5
15	100	10.1	95.9
37 dry	90	27.8	75.3

Relation of Tolerance to Pressure: In table 14 the time of exposure in minutes before the end-point of the same 14 subjects is shown from 50 ft. to 100 ft. in the wet. The geometric mean for each depth and its logarithm are given.

TABLE 14

DEPTH IN WATER	50 FEET	60 FEET	70 FEET	80 FEET	90 FEET	100 FEET
DIVER						
Kirk	3	2	1.5	3	2	2.5
Mulberry	11	15	4	4.5	4	3
Wallis	99	24.5	10.5	8.5	4	3
Robertson	35.5	19.5	11	9.5	11.5	4
Ward	35	8	38	13	7.5	3.25
Sims	26.5	13	12	13	9.5	7.5
McAtamney	35.5	16	39	18	9	5
Brown	44.5	19	14	35.5	12	11.5
Rogers	90	37	26	27.5	11.5	7.5
Whittington	59.5	73.5	19.5	8	11	12.25
Dickie	121	12.5	55	28	9.5	24.75
Smith	85	80	43.35	13	71	7.5
Fraser	120	37.5	74	44.5	18.5	19
Derrick	90	40	77.5	20	38	36.5
Geometric Mean	54.00	20.17	19.56	13.72	10.39	7.41
Logarithm of Mean	1.7324	1.3047	1.2913	1.1373	1.0165	0.8698

If these means are plotted against the depth in atmospheres absolute on double logarithmic co-ordinates then a linear relationship is demonstrated which is expressed by the equation (see Fig.13).

$$y = 3.18 \times^{-3.82}$$

where y = time in minutes

x = depth in atmospheres absolute

If the time is expressed in hours the equation is

$$y = 1.4 \times^{-3.82}$$

SHOWING LINEAR RELATIONSHIP BETWEEN LOGARITHM OF CRITICAL TIME OF EXPOSURE AND LOGARITHM OF OXYGEN TENSION

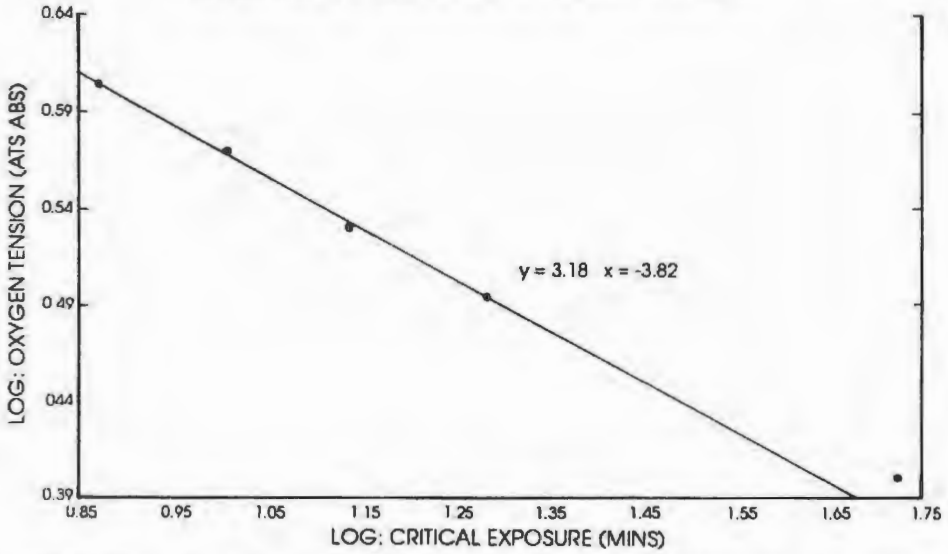

FIG. 13: The times plotted are the logarithms of geometric mean of the performances of the same 14 divers employed at all depths (see fig. 12)

Variability: Table 15 (see Appendix 2) represents an attempt to estimate the degrees of variability of the individual divers. The logarithm of the mean value for each depth is subtracted from the logarithm of the diver's time. A figure is thus obtained showing the diver's inferiority, or superiority, to the average of each depth. The variability of each diver can thus be calculated assuming that his deviation from the mean is independent of the depth. On this assumption only 40% of the total variance of oxygen divers is accounted for by the day to day variation of each individual diver. The other 60% is due to variation between the averages of different divers.

Toxicity of Oxygen in the Wet

A measure employed in the assessment of the toxicity of drugs is the concentration of the drug multiplied by the time of exposure to that concentration. In most cases a constant is subtracted from the dosage to allow for the maximal non-toxic concentration. The maximal non-toxic concentration of oxygen has not yet been determined. Here it has been assumed to be 0.209 of an atmosphere, that is the concentration in which normal physiological existence occurs. If the concentration of oxygen is

expressed as its partial pressure, in atmospheres absolute, and this is multiplied by the time of exposure, measured in hours, then the following dosages in atmosphere-hours are required to eliminate 25%, 50% and 75% at various depths.

TABLE 16

DOSAGES OF OXYGEN IN ATMOSPHERE – HOURS TO ELIMINATE 25%, 50% AND 75%

	50 feet	60 feet	70 feet	80 feet	90 feet	100 feet
25%	0.650	0.587	0.558	0.481	0.435	0.207
50%	0.939	0.870	0.921	0.789	0.588	0.363
75%	1.70	1.43	1.67	1.25	0.88	0.668

This table shows that a far smaller dosage is needed to eliminate, say, 50% at 100 ft. than at 50 ft. These results are plotted in Fig. 14.

DOSAGE OF OXYGEN TO ELIMINATE FIXED PERCENTAGES OF A GROUP

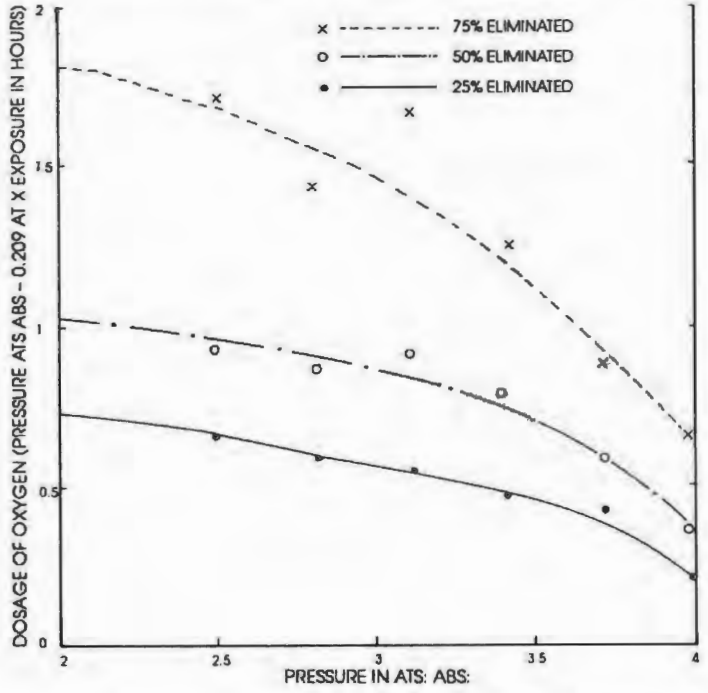

FIG. 14: Showing the decreasing dosage of oxygen (tension x time) necessary to eliminate fixed percentage of a group as depth increases.

There is obviously not only an increase in the partial pressure of oxygen as the depth increases, but a marked enhancement of the toxicity of the gas.

Oxygen Toxicity in the Wet and in the Dry

Finally, we are now able to compare the oxygen tolerance in two randomly selected groups at 3.73 ATA (90 fsw) in the wet (n = 14) and in the dry (n = 36). The greater the tolerance in the dry is clearly demonstrated in Fig. 15. (see also Tables 1 and 11).

SURVIVAL OF GROUPS OF INDIVIDUALS BREATHING OXYGEN AT 90 FEET (3.73 ATS ABS) IN THE DRY AND THE WET.

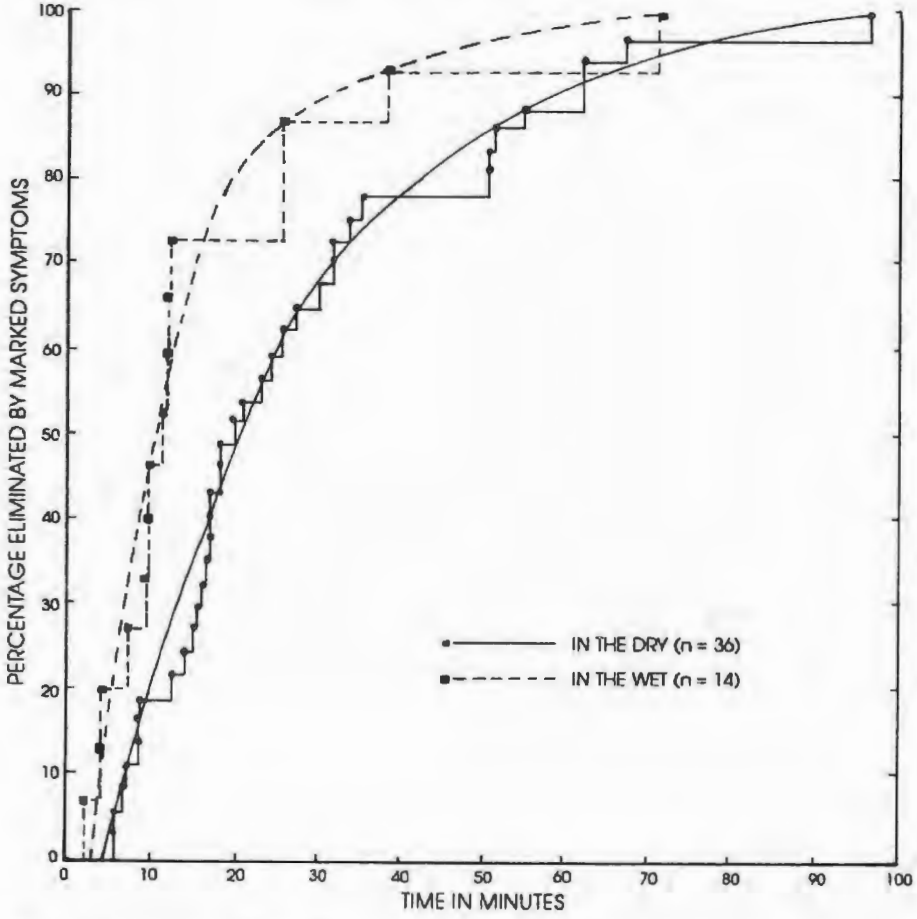

FIG. 15: Percentage of subjects eliminated by toxic symptoms at 90 feet (3.73 ATS: ABS:) breathing oxygen in compressed air and under water (65°F) in diving suit. End points plotted. No work performed during dives. (see tables 1 and 10)

Maximum Non-Toxic Depth when Breathing Oxygen Under Water at Rest

In this series of experiments an attempt was made to assess the safety of men breathing oxygen at more shallow depths and to determine at what pressure oxygen ceases to cause toxic nervous symptoms that would make free diving dangerous. As work is thought to impair oxygen tolerance, a series of dives was first carried out without exercise from 40 to 25 fsw.

Results: The individual results of these dives from 40 to 25 fsw are presented in Tables 17 to 20 in Appendix 1. For the reader's convenience a brief tabular abstract is given in the text (Table 17-20) L.T. = Lip twitching.

TABLE 17-20

Resting Dives on Oxygen to 50, 40, 35, 30 and 25 fsw

DEPTH	TIME LIMIT (mins)	NO. OF DIVERS	NO. OF DIVERS CONVG	TIME OF ONSET OF CONV. (mins)	NO. OF DIVERS WITH L.T.	TIME OF ONSET OF L.T.	NO. AND %WITHOUT SYMPTOMS.
50	120	40	16	12 to 69	21	3 to 112	3 (7%)
40	120	29	4	12 to 28	11	19 to 92	14 (48%)
35	90 (to 180)	21	1	30	5	13 to 32	15 (72%)
30	90 (to 120)	20	2	43 to 48	1	24	17 (85%)
25	120	29	0	0	1 (see text)	44	28 (96%)

It will be seen that convulsions and other toxic symptoms occur at 40, 35 and 30 fsw. 28 out of 29 divers had no symptoms during a 120 minute dive at 25 fsw. The exception was a diver who suffered nausea and vomiting after 44 minutes. This subject had similar symptoms although not so severely after approximately the same time when breathing oxygen at atmospheric pressure. Nevertheless it is possible that this diver had oxygen poisoning at 25 fsw. More prolonged exposures might have caused symptoms or even convulsions at this depth but this period of time (120 minutes) is longer

than any practical dive on oxygen to this depth. There is no statistical analysis or plotting of these results as a number of these divers were specially selected for this series because it was known that their average oxygen tolerance was poor.

It is a most surprising finding to obtain oxygen convulsions at as low a pressure as two atmospheres absolute (1.9 to 2.1 ATA). At such a tension the oxygen dissolved in the blood plasma is inadequate for even basal metabolic requirements and haemoglobin is still being actively employed for oxygen transport. Thus it appears that acute oxygen poisoning can occur before the haemoglobin oxygen cycle is de-activated and a further critical rise of brain tissue oxygen tension takes place.

The Effect of Work on Oxygen Tolerance Under Water

It has generally been considered that exercise diminishes human tolerance to hyperbaric oxygen (1943). Reference is often made to Bornstein's experiment in 1912 when he suffered clonic spasms of the arms and legs after 51 minutes exercise on a bicycle ergometer while breathing oxygen at 3 ATA in the Elbe tunnel. He had previously breathed oxygen while resting at 3 ATA for 48 minutes without ill effect. It is possible that his symptoms after exercise were due to carbon dioxide accumulation in his respiratory circuit.

Recently Behnke (1942) described how subjects breathing oxygen at 3 ATA in the dry and exercising on a bicycle ergometer suffered extreme fatigue, numbness of the lower extremities and 'abortive' convulsive seizure after 15 to 20 minutes. Usually 3 or more hours of oxygen breathing can be tolerated at this pressure.

However there are no controlled studies of the effect of work on oxygen tolerance. A large series of dives were therefore carried out in the wet with hard arm work. Control exposures with the same diver resting were also done. The subjects worked vigorously by lifting a large bag of weights by pulley without mechanical advantage. The number of lifts was measured and the height of lift controlled. Correction was made for water displacement by the weight. Dives were carried out to 50 fsw (n = 65), 40 fsw (n = 46), 30 fsw (n = 14) and 25 fsw (n = 18).

Results: These experiments show conclusively that oxygen tolerance is markedly decreased by hard work. Further, symptoms of oxygen toxicity

now definitely occured at 25 fsw. Detailed results are given in Tables 21 to 24 in Appendix 2. The amount of work performed and details of the control resting dives are also given. The impairment of oxygen tolerance by exercise can be clearly seen in figures 16 and 17 where the percentage of each group eliminated by toxic symptoms at 50 and 40 fsw, with and without work, is plotted against time in minutes to end-point.

EFFECT OF WORK ON OXYGEN POISONING IN THE WET AT 50 FEET OF SEA WATER (2.52 ATS: ABS:)

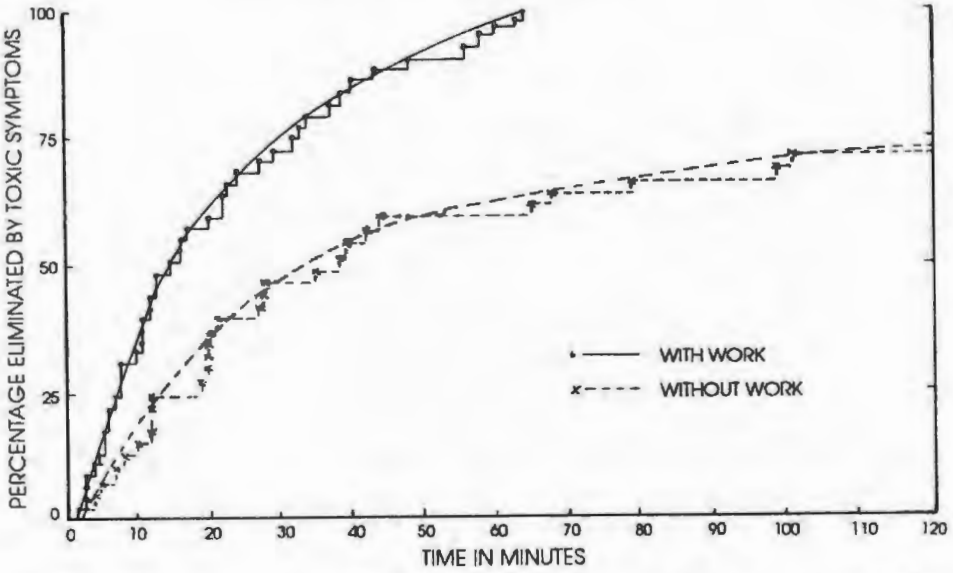

FIG. 16: Showing percentage eliminated by toxic symptoms at 50 feet in the wet (2.52 ATA) during a period of 2 hours, with and without work. Individual end points plotted. Group of 46 divers working and 41 not working. Temperature throughout 65°F.

It will also be noted in Table 24 that toxic symptoms necessitated cessation of the exposure in 5 out of 18 dives to 25 fsw in marked contrast to the resting control studies at this depth. There were no convulsions.

The physiological reasons why work reduces oxygen tolerance are not known at present. A rise of alveolar carbon dioxide, which is probably considerable when exercising on oxygen, would certainly cause cerebral vasodilatation, an increase of cerebral blood flow and of brain tissue oxygen tension, particularly as the oxygen consumption of the brain is unaltered during physical exertion.

EFFECT OF WORK ON OXYGEN POISONING IN THE WET AT 40 FEET SEA WATER (2.21 ATA)

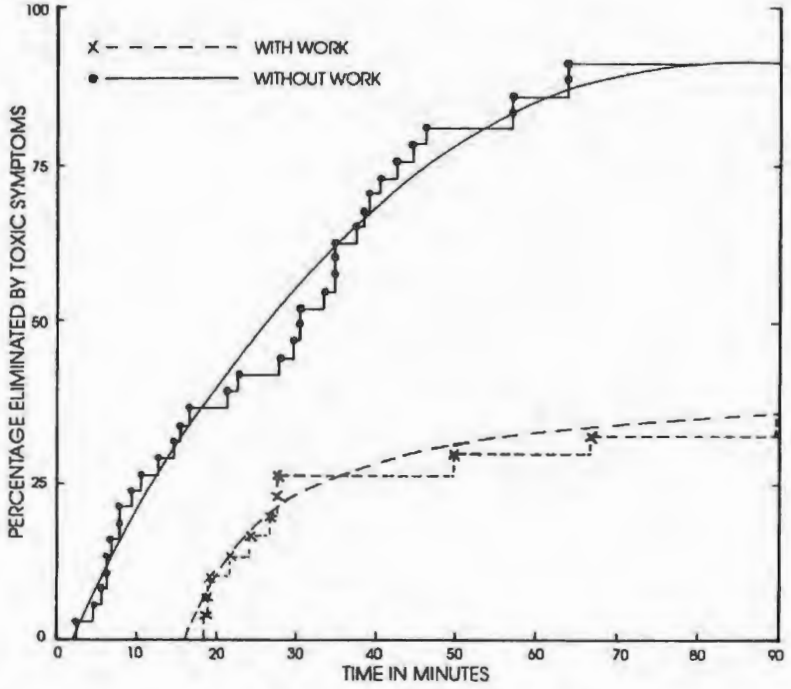

FIG. 17: Showing percantage eliminated by toxic symptoms at 40 feet in the wet (2.21 ATA) during a period of 90 minutes with and without work. Individual end points plotted. Group of 39 divers working and 31 not, working temperature throughout 65°F.

The Effect of Water Temperature on Oxygen Poisoning

Previous work has indicated that lowering of the environmental temperature increases the oxygen tolerance of small experimental animals (de Almeida, 1934 and A. Campbell, 1937).

A series of dives were carried out, in the wet, wearing the suit and apparatus used in previous experiments, to determine whether oxygen tolerance was affected by the temperature of the water in which the diver was submerged. Dives were carried out by the same group of subjects at 87.5°F (steam heated) and at 45° (ice cooled). Control dives were also performed at 65°F, the standard temperature employed in experimental oxygen dives. The group of subjects employed were on the whole of poor average oxygen tolerance. All dives were carried out at 50 ft. (2.5 ATA), this moderately shallow depth being the best at which the effect of

various factors can be assessed. Divers were allowed to vary their underwear with the temperatures, wearing light overalls in the hot series, and woollens in the cold series. It was realised that this would assist the diver in maintaining normal body temperatures but the application of this data to practical diving was the first consideration. In any case, it is very much doubted whether these experiments could have been tolerated without this variation of clothing. The gas cylinder was carried by the diver and therefore the oxygen supplied was at the temperature of the water. No exercise was carried out as this factor is known to impair oxygen tolerance. This was advantageous at the higher temperature, but disadvantageous to the diver in cold water. The majority of divers complained of the cold bitterly and found the heated dive very uncomfortable. Although body temperatures were not obtained, symptoms reported would suggest that these were affected to a certain degree towards the end of the dive.

Results: These are shown in the following table (25) and Fig. 18.

THE EFFECT OF TEMPERATURE OF WATER ON OXYGEN TOLERANCE IN MAN

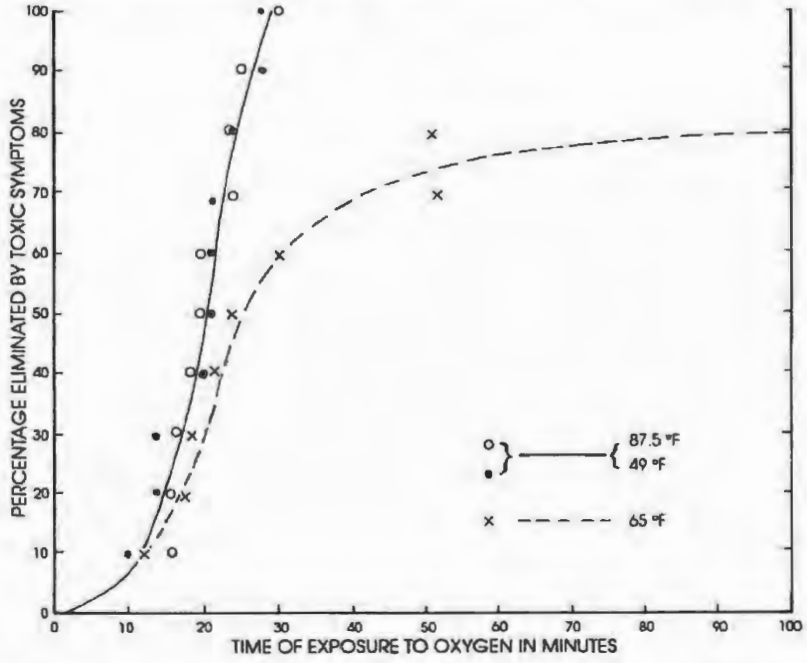

FIG. 18: Showing percentage eliminated by toxic symptoms at a pressure of 50 feet of sea water (2.51 ATA) in the wet at 65°F, 87.5°F and 49°F, plotted against time of exposure. Subjects resting.

TABLE 25

VARIATION OF TEMPERATURE. ALL DIVES AT 50 FT. TIME IN MINS.

Temperature	65° F		87.5° F		49° F	
Name	Time	Symptoms	Time	Symptoms	Time	Symptoms
Knight	17	Severe lip twitching	24	Lip twitching	21	Severe lip twitching
Witham	22	Severe lip twitching	16	Lip twitching	14	Lip twitching
McInnes	100	None	29	Lip twitching	20	Lip twitching
McCann	30	None	17	Lip twitching	20	Lip twitching
Gibson	12	Convulsed	23	Convulsed	10	Lip twitching
Gray	51	Lip twitching	25	Lip twitching	21	Lip twitching
McLaughland	24	Convulsed	16	Severe lip twitching	27	Lip twitching
Green	18	Lip twitching	20	Lip twitching	27	Lip twitching
Murton	102	Nausea	20	Headache	14	Lip twitching
Shields	51	Lip twitching	20	Lip twitching	24	Lip twitching

It is apparent from these results that the oxygen tolerance is somewhat impaired by both cold and heat.

It will be noted that, although the averages of performance in the hot and in the cold, are about half of that at 65°, the medians of time of tolerance are approximately similar at all temperatures. A possible explanation of this is that body temperatures were not greatly disturbed until after submersion of about 20 minutes duration.

Signs and Symptoms of Oxygen Poisoning

Convulsive Attacks: These convulsive attacks were, on average, of two minutes duration, the diver being unconscious. Incontinence occurred in only 5% of cases. If the subject was turned on to air immediately convulsive symptoms commenced, only one attack resulted. The subject remained

unconscious and flaccid for five to ten minutes after the convulsion. Breathing was stertorous and the cough reflex absent. The pulse was accelerated and bounding. The corneal reflex usually returned within about three to four minutes. The eyes which were averted, then returned to the central position when a rapid horizontal nystagmoid movement was usually to be seen. The pupils were dilated and insensitive to light but, shortly before consciousness was regained, they assumed their normal size and reacted to light. About this time the cough reflex returned. Subjects, on regaining consciousness, were markedly confused and dissociated and in some instances, extreme emotional instability was exhibited. Laughter, tears, singing and extreme violence were all encountered. The majority were subdued, dissociated, and ataxic for about 15 minutes afterwards. The degree of ataxy varied. In a marked case the stance was wide and the stepping act faulty, the foot either being lifted excessively or hardly at all. Gersh, (1944) in work carried out since these experiments, describes an obviously associated phenomenon in cats which lost their stepping and hopping reflexes after a number of oxygen exposures to convulsion. These animals recovered after several weeks. It would appear that the same nerve cells are affected, even after one convulsion, in man although recovery is very rapid.

Most subjects complained of headache and some had nausea and vomiting with associated anorexia. Photophobia was reported by some of the subjects. Fatigue was usually marked and the natural tendency was to "sleep it off" for about half an hour. In a few cases there was post-convulsive automatism, the subject being quite rational in conversation and behaviour, but waking up the next morning with complete amnesia of the period since the convulsion. For this reason subjects were not allowed to be alone after a convulsion for a period of twenty-four hours when normality was always regained.

These convulsive attacks, in fact, were clinically indistinguishable from an idiopathic epileptic convulsion; the tonic phase, however, did not last for more than one or two seconds. No permanent residue of any description have been found in this series which have lasted over a period of three years. There were some stiff backs and subcutaneous extravasation due to muscular violence. One subject, who had convulsed twice, developed petit-mal about six months later. A strong family history, which had been previously concealed, was elicited.

Warning Signs and Symptoms: Practically every symptom described in the aura of idiopathic epilepsy has been encountered during the period before the onset of the convulsion. Auditory hallucinations such as music, bell ringing and knocking, flashes in front of the eyes, impaired vision, unpleasant tastes and odours and paraesthesia have all been reported. Muscular twitching, sweating, vertigo, nausea, apprehension, euphoria and dreamy states have all occurred. These warning symptoms are of great importance as an accurate knowledge of such symptoms may enable persons who are exposed to dangerous tensions of oxygen to discontinue before convulsions occur. Over two thousand dives have been carried out breathing toxic tensions of oxygen, about 400 of these have been in the dry and the rest in diving suits under water. Consequently a much larger and complete picture of the signs and symptoms of oxygen poisoning in the human subject has been obtained.

First let us consider the symptom of oxygen poisoning when breathing oxygen in a dry pressure chamber. Such experiments allow the diver and observer more opportunity for introspection and observation, respectively. It is not easy to give a clear and concise description of human symptoms under toxic tensions of oxygen. There is enormous variation of the syndrome in different subjects. First, three specific cases are described to give some idea of the different events encountered.

SUBJECT A: 101 minutes of oxygen at 60 fsw (2.8 ATA). Facial pallor, almost at once, which continued throughout the dive. Fine fibrillary tremors of lips noted after a few minutes. These came and went throughout the dive and were occasionally seen in the upper face. Subject complained of drowsiness during the last twenty minutes. A few seconds before the end he had severe twitching of the lips; the nose was also involved. Severe diaphragmatic spasm just before coming on to air.

SUBJECT B: 25 minutes at 90 fsw (3.7 ATA). Facial pallor marked during whole exposure. Passed into tonic phase of convulsions without any warning signs or symptoms.

SUBJECT C: 54 minutes at 90 fsw (3.7 ATA). Facial pallor not marked till after about twenty minutes. Choking sensation after thirty

minutes, which passed off. Slight but definite fibrillation of upper lip after eighteen minutes which continued for the next twenty-five minutes. Some definite and sustained lip twitching four minutes from the end and severe generalised lip twitching at the end.

These are only three random examples of a very varied picture. An attempt will be made to survey the general findings.

Facial pallor usually occurs a few minutes after the commencement of the exposure. It varies from person to person in degree and time of onset and may be generalised or circumoral. The general appearance of the face after this presumable skin vaso-constriction naturally varies. A number of subjects appeared like patients with mitral stenosis. One subject had a large recently vascularised scar on the face. The whole of his face appeared dead white, apart from the scar, which stood out a most conspicuous red, presumably because the newly vascularised tissue had not the same degree of vasomotor control. The degree of facial pallor is no indication of the subjects sensitivity nor of an impending end-point. In a large number of cases very fine fibrillations of the lips or face may be seen early in the experiments and intermittently throughout the exposure. These were attributed to two factors, the fatigue of the lips holding the mouthpiece and a very natural nervous tension. Some subjects showed such fibrillations when wearing a mouthpiece at atmospheric pressure but they are undoubted increased by breathing oxygen at increased tensions. Fibrillation frequently appears in muscles which later show severe and sustained twitches. A number of subjects showed facial perspiration varying in degree from fine beads on the upper lip and forehead to literal pouring. Generalised perspiration is not usual but is occasionally seen. Slight nausea and vertigo and choking sensations that wax and wane may be encountered some time before the end-point. The course of these minor crises is absolutely unpredictable and the observer has to be constantly on the alert for a sudden exacerbation of symptoms with the danger of convulsing.

The next group of symptoms to be discussed may be called warning symptoms which, although not demanding immediate cessation of the exposure, signify that intoxication is becoming more intense and that the end-point will not be greatly delayed. Sensations of sleepiness, depression

or euphoria are encountered in a number of cases about 10 minutes (at 3.7 ATA) before the end-point. Apprehension may be marked and increase as the end-point approaches. Changes in behaviour may be noted by the observer during this period; the subject showing clumsiness with his apparatus, loss of balanced judgement, fidgeting or an unnatural disinterest in the experiment. Sensations of oppression in the praecordium are described during this period. Palpitations, which may involve not only an awareness of the heart's action but of the arterial pulsation throughout the body, are occasionally reported. Visual symptoms include loss of visual acuity, dazzle in the visual field, lateral movement of visual images and apparent changes in the intensity of illumination. Subjects have described the constriction of the peripheral visual field without knowledge of this phenomenon. This is encountered more frequently after prolonged exposures at toxic pressures and it is therefore only experienced by the more resistant subjects. Acoustic hallucinations are not common. Music, bell ringing and knocking have all been reported. No voices have been heard. On other occasions hearing may become impaired. Nausea and vertigo, together or separately, may become so severe that the subject will revert to air breathing before convulsive symptoms occur.

Definite twitching movements of the lips usually mean that the end-point is not far off. This is the most common end-point. The twitches are powerful and sustained. They are usually seen one side of the upper lip but if the exposure continues they increase in power and frequency and spread to the whole mouth and face. On occasions a marked twitch may be followed by a long period of quiescence before recurrence. This is exceptional and in most cases, if the exposure is continued, convulsive movements of the lips pass into generalised jactitations or far more commonly, convulsions. Twitching of the cheek and nose are often seen with or without lip twitching. Occasionally isolated twitching of the arm or leg will occur.

In a few minutes or seconds before the end-point, respiration, which in most cases has been normal and serene throughout, shows a number of abnormalities. The commonest occurrence is rapid panting. In other cases there appears to be a marked inspiratory predominance probably associated with tonic contraction of the diaphragm. This may give rise to a grunting respiration, to a sensation of abnormality in the epigastrium or, in the more severe case, to an acute state of apnoea in the inspiratory

position. Hiccough is another symptom of diaphragmatic disturbance. On being turned onto air many subjects recover but, with less fortunate subjects, it may be too late and they pass into convulsions.

Three unusual end-points can be mentioned. In one case, after about ten minutes of vertigo and dazzle, the subject commenced agonising, uncontrollable, spasmodic vomiting which continued for several minutes. In another case the subject passed into a heavy, stuporous sleep from which he was roused with difficulty. In the third case acute syncope occurred with pallor, cold sweat and thready pulse. The clinical picture was indistinguishable from that of acute traumatic shock.

As can be seen from the above, an acute description of such a varying picture is not easy. The impression gained is of two distinct processes occurring in may different patterns. One is an insidious intoxication which may affect the function of practically any part of the central nervous system and, added to this, is an increasing convulsant tendency which is usually, but not always, first manifested in the facial muscles and finally becomes generalised. There are great variations in the resistance of the individual to the general background of intoxication and in the resistance of the individual to the convulsant factor. Again, certain individuals may show powerful convulsive movements, either localised or generalised, but retain consciousness. Others pass into what is indistinguishable from an epileptic fit immediately after such convulsive movements and, occasionally, in their complete absence.

Off Effect: The "off effect" on returning to air breathing after exposure to toxic tensions of carbon dioxide is well known. There is a sudden exacerbation of symptoms often associated with headache, nausea and vomiting. An "off effect" is sometimes encountered after the cessation of breathing oxygen at toxic tensions. It does not occur so frequently as that after breathing carbon dioxide. Severe nausea, increased pallor, sweating and vertigo may all occur in a subject who was previously symptomless. Other subjects show a sudden marked dissociation and panting. In one or two cases it appeared that convulsions were precipitated by reverting to air breathing. This may be due to the fact that such a degree of toxaemia had been reached that convulsions were inevitable, although reversion to air breathing had taken place. A possible explanation of the oxygen "off effect" may be as follows. It is known that the respiration of the brain tissue is impaired by high tensions of oxygen. As a result of Dicken's work,

it appears not unlikely that the nerve cells are "eliminated" individually and show a distribution of tolerance very similar to that demonstrated in a group of men. It is possible that certain cells, whose elimination will cause symptoms or convulsions, are on the brink of this event. The sudden reduction of oxygen tension to a far lower non-toxic level may, by the law of mass action, further reduce metabolism in these damaged cells and the resultant cessation of function cause symptoms or even convulsions. This theory is favoured by the fact that a number of subjects with a severe end-point have convulsed during decompression in air, when the oxygen tension is lowered even further. It has therefore become a rule in this work that, if a subject has severe symptoms and has reverted to air breathing, he is not decompressed until his symptoms have remitted and relative normality has been attained. In experimental work with animals in high tensions of oxygen a number of observers have noted that several animals, which were previously symptomless, began to convulse during decompression. If the pressure is very great and the decompression very rapid this may be due to bubble formation but these convulsions usually occur long before sufficient fall of pressure has taken place for such an explanation to be valid.

After Effects with Non-Convulsant End-Point

The recovery from a non-convulsant end-point is remarkably rapid and comparative normality is attained in five, or less, minutes. Twitching usually ceases in about a minute or less. The subject often appears dazed for a few minutes longer and his respiration are irregular with intermittent deep excursions. Euphoria is frequent but this may well be due to a natural relief at having survived a toxic exposure without convulsing. Pallor persists in some cases for as long as an hour and in a few cases, the subject behaves as if he is slightly intoxicated for the same period. This latter syndrome is known in experimental diving circles as "oxygen jag".

> *The most important finding in this large series of exposures was that the symptoms of oxygen poisoning vary enormously in different people and in the same person during different exposures. No list of warning signs or symptoms can be given that would ensure a safe and timely cessation of the exposure.*

The traditional symptom of paraesthesie of the extremities is very rare there being only one case in five hundred toxic end-point exposures.

Oxygen Poisoning Under Water

In a series of 388 non-working dives to end-point, under-water symptoms were recorded and classified. Results were as follows:

TABLE 26

Symptoms	Number of Cases	Percentage
Convulsions	46	9.2
Twitching of lips	303	60.6
Vertigo	44	8.8
Nausea	43	8.3
Respiratory Disturbances	19	
Dyspnoea	8	
Coughing	6	
Spasmodic respiration	2	
Diaphragmatic spasm	2	
Choking sensation	1	
Twitching of parts other than lips	16	3.8
Generalised jactitations	7	
Upper face alone	2	
Arms	2	
Legs	2	
Hands	1	
Palate and fauces	1	
Trunk muscles (erector spinae)	1	

Symptoms	Number of Cases	Percentage
Sensations of Abnormality	16	3.2
Drowsiness	7	
Numbness	3	
Exhaustion	3	
Malaise	2	
Confusion	1	
Visual Disturbances	5	1
Dazzle	3	
Loss of vision	2	
Acoustic Hallucinations	3	0.6
Paraesthesiae	2	0.4
Generalised	1	
Facial	1	

The most striking observation was the remarkable predominance of lip twitching as the only symptom. It is probable that many of the other more subtle symptoms occur, but that they are difficult to appreciate in a diving suit under water.

Symptoms in the Wet with Work

It must be emphasised that throughout the series in which symptoms have been described the subjects were not carrying out exercise. Since Bornstein's single experiment in the dry on an ergometer (1912), it has been stated in the literature that exercise at toxic depths causes twitching of the muscles being employed.

During the series at toxic depths with hard arm work, analysis of symptoms in 120 end-points gave the following figures:

TABLE 27

Symptoms	Number of Cases	Percentage
Convulsions	7	6.8
Lip Twitching	60	50
Vertigo	25	20.8
Nausea (vomiting in two cases	21	17.5
Choking sensation (pharyngeal spasm)	3	2.5
Dyspnoea	3	2.5
Body tremors	2	1.7

It appears that nausea and vertigo increase in frequency, if the subject is exercising. The most interesting finding, however, was that twitching of the muscles being exercised was not encountered in the whole series. These divers were carrying a very large and efficient absorbent canister and as already suggested, the symptoms described by Bornstein may have been due to added carbon dioxide poisoning. A series of experiments carried out to investigate the effect of work on oxygen tolerance in the dry were marred by inadequate carbon dioxide absorption and are not reported here.

Behaviour of Subject Before Toxic Symptoms Appear

Divers breathing oxygen at increased tensions, before signs or symptoms of toxaemia occur, feel remarkably normal. The mental torpidity, described by some observers, has not been noted even at considerable depths except after long exposures in the dry. Men have been able to carry out complicated tasks necessitating high skill and judgement while breathing oxygen under water at markedly toxic pressures. The capacity for hard physical work appears to be in no way impaired. Underwater divers are more free of symptoms than those in the dry, right up to the moment of lip twitching or convulsing. No doubt the abnormal environment and accoutrement obscure minor premonitory symptoms.

Divers who have gone on to oxygen at such depths as 100 ft., have

volunteered the information that they felt an immediate clarification of thought. This is due to the sudden cessation of exposure to increased tensions of nitrogen. The normality of the subject breathing oxygen at these tensions and the frequent suddenness of convulsive symptoms makes oxygen breathing at toxic depths highly dangerous, particularly as the subject often gains a very false sense of security.

The "Lorrain Smith" Effect in Divers

Information on this subject is very scanty. In the experiments described by Behnke, Johnson, Poppen and Motley (1934/35) four subjects breathing oxygen in the dry at three atmospheres absolute for three hours, four subjects breathing oxygen in the dry at three atmospheres absolute for three hours, and two subjects breathing oxygen at four atmospheres for 40 minutes, showed no signs or symptoms of pulmonary damage.

In view of the fact that oxygen appears more toxic when breathed under water, as judged by the onset of nervous signs and symptoms, it was considered possible that the pulmonary effects might also be accelerated. No data is available concerning lung damage in divers breathing raised tensions of oxygen under water. The series in the dry, mentioned above, is far too small for general conclusions to be made.

In well over a thousand experiments, where subjects were breathing oxygen at toxic pressures (4.68 to 1.91 atmospheres absolute) the exposure was terminated owing to signs or symptoms involving the central nervous system. No signs or symptoms of pulmonary irritation or damage were encountered during the whole series. Frequent chest examinations were completely negative. A few examples of long dives breathing oxygen, in the wet and dry, are here given:

IN THE DRY (Time mins/Press. ATA)		IN THE WET (Time mins/Depth fsw)	
Whittington,	159/2.8	Warren, Dickie, Fraser	120/50 (2.5 ATA)
Harman,	180/2.8	Fraser,	45/79 (3.4 ATA)
Whittington,	55/3.7	Miller,	75/79 (3.4 ATA)
Harman,	62/3.7	Derrick,	37/99 (4 ATA)
Derrick,	96/3.7		
Smith,	61/4.7		

It was concluded from this very large study of oxygen dives, that at toxic tensions, nervous symptoms or convulsions will terminate the exposure before any demonstrable harm is suffered by the pulmonary epithelium. At more shallow depths, however, nervous symptoms are only encountered after very long exposures or not at all. It was thought possible that there may be a greater risk of lung damage at such depths. Dives for three hours at 2.1 atmospheres absolute (36 fsw) in the wet caused no pulmonary irritation. A series of prolonged dives to 12 ft. (1.36 atmospheres absolute), with periods at 50 ft. (2.5 atmospheres absolute), gave equally negative results. Incidentally none of these divers had any nervous symptoms.

Prolonged Dives: These divers cruised or exercised in a leisurely fashion at 12 feet. There was no significant exercise in the deeper excursions. (Time in mins./Depth fsw).

Derrick (i)	–	180/12, 30/50, 90/12 5 hours continuously on oxygen at 12 and 50 fsw.
Derrick (ii)	–	120/12, 30/50, 120,12, 30/50 5 hours continuously on oxygen at 12 and 50 fsw.
Rickwood	–	240/12, 30/12 5 hours continuously on oxygen at 12 and 50 fsw
Goss	–	249/12, 30/50, 90/12 6 hours 9 minutes on oxygen at 12 and 50 fsw

It can be stated, with reasonable certainty, that no real underwater dive will ever be made where lung damage will result from high tensions of oxygen.

With regard to comparison with animals, L. Smith (1899) reported rats dying of pulmonary damage as early as 20 minutes on oxygen at 4.5 ATA. Yet the subject Smith completed 61 minutes at 4.7 ATA in the dry with no symptoms or demonstrable pulmonary damage. Total evidence available at present suggests that man has more resistance than small experimental animals to lung damage by high tensions of oxygen.

Cumulative Effects

A number of these subjects have breathed oxygen at increased tensions several times a week for two years. It was considered possible that, although the pulmonary damage suffered in a single exposure was inappreciable, there may be a cumulative effect. Frequent routine examinations of the subjects' chests were therefore carried out, x-rays were taken regularly and the vital capacity noted. In not a single case has there been any positive finding. Their general health and considerable athletic activities have also been carefully watched and again, with negative results. One subject who dived to toxic depths to end point two or three times a week for three months won the Portsmouth Middle Weight Boxing Championship during this period. The weight records of all subjects have been satisfactory. It would appear that there is no cumulative effect on the lungs in oxygen diving.

Cardiovascular Findings in Oxygen Poisoning

It was amply confirmed that breathing oxygen at increased tensions (2 to 4 ATA) caused bradycardia in the majority of cases.

The degree of this occurrence varies enormously. Subjects were resting for 15 minutes before the exposure and the prolonged rest and quiet environment may have caused further slowing. The initial pulse rate may also have been raised owing to "keying up" before the exposure. A number of subjects had symptoms which could well have caused increase of the pulse rate, as a result of apprehension, but the slowing of the pulse continued. The degree of bradycardia had no fixed relation to tolerance nor did the pulse changes give any warning of acute symptoms. Summarising, it can be stated, although the pulse changes in a number of cases were little more than can be accounted for by prolonged basal conditions, in other cases a marked and definite bradycardia was encountered.

Only a limited number of blood pressure recordings were made at 90 ft. There was a gradual slight rise of systolic and diastolic pressure for 20 to 25 minutes. Diastolic pressures then remained steady at 10 to 15 mm. higher than normal, except for a few minutes before the end-point when a further brisk rise occurred. These findings are similar to those of Behnke, Forbes and Motley (1935/36). In the shorter exposures the 'terminal' rise was obtained before the blood pressure had stabilised at its new level.

A number of subjects' nail bed capillaries were observed microscopically while they were breathing toxic tensions of oxygen. Controls were carried out breathing air at atmospheric and increased pressures and oxygen at atmospheric pressure. No significant changes were found. One subject's capillaries were under observation while suffering from lip twitching and no changes were seen.

Facial pallor has already been described in the section on symptoms.

X-ray and clinical examination have shown no enlargement of the heart in subjects who have been frequently exposed to high tensions of oxygen over a long period. It is a common belief among divers (1942), particularly in the Italian Navy, that oxygen breathing causes increase in the size of the heart. This has not been confirmed.

Neurological Findings in Increased Tensions of Oxygen

Routine neurological examination of subjects breathing oxygen at 90 and 60 ft. (3.73 and 2.74 atmospheres absolute) showed no significant change in reflex activity. Restriction of the peripheral visual field was described by a number of subjects but specific tests were not carried out. The only other finding was the development of a positive Chvostek sign (twitching of face on tapping the facial nerve in front of the external auditory meatus) in a number of subjects during exposures to hyperbaric oxygen in the dry. This sign usually developed in the latter half of the exposure; it was not a reliable sign of the approach of acute symptoms although it became more marked as the exposure proceeded. Some subjects had an acute end-point without the occurrence of a positive Chvostek sign at any time. Controls carried out in air at atmospheric pressure, revealed that a number of otherwise normal subjects have a positive Chvostek sign which may be present one day and absent on another. In one experiment a subject developed a unilateral positive Chvostek sign while breathing oxygen at atmospheric pressure. It disappeared a few minutes after the cessation of the exposure. A positive Chvostek sign in air, or developing during the exposure, did not necessarily signify poor oxygen tolerance.

Jacksonian Attack: One very interesting subject can be mentioned here. This man had a number of severe end-points including two convulsions. Acute toxic nervous symptoms always commenced with twitching of the muscles of the left hand which then spread to the arm. Lip twitching was

usually present at this stage. The subject then convulsed or recovered on being turned on to air. The series of events, in fact, were similar to those in a Jacksonian epileptic attack and would suggest that this subject had a slightly lower threshold in one small cortical or sub-cortical area that precipitated the convulsive process. There was no known history of head injury or relevant disease.

Neurological Residua of Oxygen Poisoning and Convulsions

This question is of great importance in human physiology. The convulsant action of oxygen has been suggested by several authorities for the therapeutic treatment of psychoses (1945). In this series of experiments every attempt was made to avoid convulsions. It can be stated that in three years of continued experiments, no evidence of any kind has been obtained to show that acute oxygen poisoning, or even several convulsions, has any adverse after effect on the person's neurological integrity, intellectual ability or personality. Such a finding is in accord with those in animal experiments. Finley could find no histological abnormality in the brains of rats convulsed till death. Gersh (1944) could demonstrate no functional or histological abnormality in cats after a single convulsion and found that many successive exposures to the point of convulsion gave such a slight histological findings that are artefact could not be excluded. The neurological findings in animals with maximal damage cleared up in a few weeks.

Electroencephalographic Findings: Note: These investigations were carried out at the National Institute for Medical Research by Brown, Downman and MacIntosh. Expert opinion on the recordings was given by Wing Commander D. Williams. Only those experiments which were carried out on the subjects in the unit are briefly described here. They are more fully reported in R.N.P.R.C. 94/1944.

Exposure to oxygen at 120 ft. had no immediate effect on the E.E.G. recording. In general, there was a slow increase in the amount of fast activity i.e. the 25-32/second band and also an increase of the voltage of the 3-5/second waves. Coupled with this was a progressive decrease of the amount and voltage of the dominant frequencies, i.e. the 8-12/second band. The tracing tends towards a sequence of 3-5/second wave with a super-imposed ripple of fast activity.

Infrequently, spikes i.e. single, high voltage, fast, sine waves appeared and increased in number shortly before the end-point. Subjects who had non-convulsive end-points showed no other changes. Those who convulsed gave a picture of electrical activity, during and after the fit, which was indistinguishable from that seen in grand-mal epilepsy. It was apparent that there is nothing specific in the convulsions of oxygen poisoning, as regards electrical activity, once they have commenced. In some cases there were signs of disturbances, i.e. short bursts of 5/second activity, with increasing voltage, just before the attack. Others showed no change of cortical electrical activity whatsoever before the major convulsive attack. In view of the similarity of the convulsion to those in grand-mal, both clinically and electrically, it was thought that a study of the E.E.G. of subjects in air, and with hyperventilation, might show inborn instabilities or convulsant tendencies that could be correlated with oxygen tolerance.

Fifteen subjects were graded in order of their oxygen tolerance. This was based on average tolerance during many dives at 60 ft., 90 ft., and 120 ft. in the dry and 50 ft. in the wet. E.E.G. records were classified as normal, abnormal or doubtful (suspicious but indefinite features present). Final assessment was the result of two independent opinions. Results are given in the following table.

TABLE 28

Subject's Endurance Rating	E.E.G.	Whether Convulsed
1	Normal	-
2	Normal	-
3	Normal	-
4	Doubtful	1
5	Abnormal	2
6	Doubtful	-
7	Abnormal	2
8	Abnormal	-
9	Doubtful	-
10	Doubtful	2
11	Normal	-
12	Doubtful	-
13	Doubtful	3
14	Normal	-
15	Doubtful	1

There is no statistically significant correlation although it will be noted that the three most resistant subjects had normal E.E.G's. However, the other two "normals" occurred in the last five in endurance rating. It is of interest to note that all those who had actually convulsed had abnormal or doubtful records. Nevertheless the third most resistant subject with a normal E.E.G. convulsed after this series was completed. Personality assessments also gave no definite correlation with oxygen tolerance.

Conclusion

In the first large series of experiments on human beings knowledge of the dangers and symptoms of oxygen poisoning has been greatly expanded. It has been clearly demonstrated that these dangers are far greater than previously realised.

> *The variation of tolerance between individuals, the variation of tolerance of each individual, the impairment of tolerance with work and under water, all make diving on pure oxygen below 25 feet of sea water a hazardous gamble.*

The impairment of tolerance under water is as mysterious as it is unfortunate. Despite the fact that the first comprehensive description of human symptoms of oxygen poisoning is given here, it is emphasised that no signs or symptoms can be given that would ensure a timely cessation of oxygen breathing in all cases. The variation of symptoms, even in the same individual and at times their complete absence before convulsions, constitute a grave menace to the independent oxygen diver.

The only possible conclusion is that such tensions of oxygen should be scrupulously avoided.

REFERENCES

ALMEIDA, A.O.de (1934): Recherches sur l'action toxique des hautes pressions d'oxygène. *C.R. Soc. Biol., Paris*, **116**, 1225-1227.

BEHNKE, A.R. (1942, (i)): Physiologic studies pertaining to deep sea diving and aviation. *Bull. N.Y. Acad. Med.*, **18**, 561-584.

BEHNKE, A.R. JOHNSON, F.S. POPPEN, J.E. and MOTLEY, E.P. (1935): The effect of oxygen in man at pressures from 1 to 4 atmospheres. *Amer. J. Physiol.*, **110**, 565-572.

BEHNKE, A.R., FORBES H.S. and MOTLEY, E.P. (1936): Circulatory and visual effect of 0_2 at 3 atmospheres. *Amer. J. Physiol.*, **114**, 436-442.

BORNSTEIN, A and STROINK, M. (1912): Ueber sauerstoff vergiftung. *Dtsch. med. Wschr.*, **38**, 1495-1497.

BROWN, G.L., DOWNMAN, C.B.B., MACINTOSH, F.C. and WILLIAMS, D. (1944): The effect of oxygen at high pressure on the human electro-encephalogram. *Royal Naval Personnel Research Committee*, 94. (UPS 39/44).

CAMPBELL, J.A. (1937): Body temperature and oxygen poisoning. *J. Physiol.*, **89**, 17-18.

DICKENS, F. (1945): Personal communication. Since published (1946).

DICKENS, F. (1946): Toxic effects of oxygen on brain metabolism and tissue enzymes. *Biochem. J.*, **40**, 145-187.

DONALD, K. W. (1944): Variation of oxygen tolerance of human subjects in the wet and in the dry. *Admiralty Experimental Diving Unit (R.N.) Report XIII* (incorporting AEDU Reports I to VI, 1942-43).

DONALD, K.W. (1945): Oxygen poisoning in man. *M.D. Thesis, Cambridge University*. Later issued as *Admiralty Experimental Diving Unit Report XVI/1946*.

GERSH, I. (1944): Oxygen poisoning. *U.S. Navy Research Project X192*.

HEDERER, C and ANDRÉ, L. (1940): De l'intoxication par les hautes pression d'oxygène. *Bull Acad. Méd. Paris.*, **123**, 294-307.

SMITH, J. LORRAIN (1899): The pathological effects due to increase of oxygen tension in air breathed. *J. Physiol.*, **24**, 19-35.

NOTE

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CHAPTER 3

FURTHER STUDIES OF OXYGEN POISONING 1946 TO THE PRESENT

In 1946 Hayter and White (US Navy) reported a quite remarkable decrease of oxygen tolerance of divers exercising very moderately on a bicycle ergometer (300 kgm/min; VO_2 0.85 1/min. approx.) in a dry chamber at 59 fsw gauge (2.71 ATA). Whereas a resting subject can breath oxygen for two to three hours without untoward effects at this pressure in the dry, these 13 exercising subjects had "typical symptoms" of oxygen poisoning in an average time of 10 minutes (range 6 to 18 mins.). Two subjects convulsed after 11 and 13 minutes, one while breathing oxygen and the other 40 seconds after removing his mouthpiece because of lip twitching. The subjects breathed oxygen from a demand system "designed to prevent rebreathing".

Behnke (US Navy), commenting on the Royal Navy reports (Donald, 1944, 1945) on oxygen poisoning, recognised "the difference in tolerance between wet diving and that in the dry chamber as a new finding". Nevertheless he still felt that divers "in the inactive state" could breath oxygen during decompression under water for considerable periods in safety (Behnke, 1946), despite the formidable evidence against this view. The US Navy continued to employ underwater oxygen decompression stages, first at 60 fsw to 40 fsw and later at 50 and 40 fsw.

Yarbrough, Welham, Brinton and Benke (1947)

This report describes the first United States Navy series of under water oxygen dives from 100 to 30 fsw under controlled chamber conditions. They confirmed the Royal Navy findings as regards wet/dry difference of oxygen tolerance, the safe limits of working oxygen dives, the variation of tolerance in and between individuals and the varying pattern of the symptoms of oxygen poisoning.

"Wet/Dry" Difference of Oxygen Tolerance: Exposures in compressed air and under water with the subjects breathing oxygen were performed at 60, 80 and 100 fsw. In the dry chamber experiments oxygen was administered by a demand system with an open circuit. In the wet chamber dives closed circuit self contained apparatus (Browne or Lambertsen type) were worn. The water temperature was high (90° F). They reported "it was found that, in 20 exposures in the dry chamber at rest at a simulated depth of 60 feet, symptoms did not occur during a period of two hours. However, at the same depth under water, at rest, 32 out of 107 exposures were terminated prior to 60 minutes. The average time of termination was 32 minutes, range 8 to 58 minutes. Two convulsive seizures occurred, one at 13 minutes and the other at 24 minutes."

At 80 fsw, in the dry, 56% of 46 subjects terminated in the first hour in contrast to 75% of 99 subjects in the wet. Interestingly, at 100 fsw the figures were closer: in the dry 88% of 26 subjects terminated in the first hour in contrast to 93% of 46 subjects in the wet. Unfortunately, apart from the two convulsive episodes mentioned above, no individual results are reported so information as to the distribution of the tolerance of these subjects is not available.

In contrast to these deeper dives at rest, all exposures at 50, 40 and 30 fsw were working underwater dives. Self contained closed circuit oxygen breathing apparatus was worn. The divers performed weight lifting arm exercises, the estimated work being 1200 ft.lbs. per minute. This was roughly of the same order of arm work as that performed in the Royal Navy exercise series. (Donald, 1945). Dives were up to a maximum of 120 minutes. At 50 feet three of five exposures were terminated before that time, the earliest end-point being at 32 minutes. At 40 feet, 11 out of 48 subjects terminated, the earliest end-point being 44 minutes. At 30 feet 1 out of 17 subjects terminated at 87 minutes. Individual end-points and symptoms were not given. The authors concluded that "variability in time of onset of symptoms in the same individual does not permit the setting of precise time limits for depths in excess of 30 feet when work is performed."

No dives were carried out at 50, 40 and 30 fsw with the diver at rest. This was an unfortunate omission in so far as the US Navy had repeatedly claimed that, despite the British findings, the resting diver had such a high degree of oxygen tolerance that they were safe using oxygen decompression stages from 60 to 40 feet in open water. Yet in the whole of

this series no comparison was made between exercising and resting divers' oxygen tolerance at the same depth. As far as the present author knows, the Royal Navy series (1944-5) of resting dives at 50, 40, 30 and 25 fsw has never been repeated.

In this US Navy study the variation in individual oxygen tolerance was studied by repeated resting oxygen dives to 60 fsw (wet chamber). Again self contained breathing apparatus was worn. Almost all exposures were terminated at 60 minutes but a few dives were extended to 120 minutes. The period between individual dives was one or more weeks. Divers showed considerable individual variation. One subject had the following series of dives. (Time (in minutes), alone, means no symptoms).

60; 60; 24 convulsed; 15 muscular twitching; 60; 23 muscular twitching.

Another had marked tolerance except for one occasion, as follows:

60; 60; 32 nausea and muscle twitching. 60; 60; 120.

Some showed poor tolerance almost consistently i.e.

(a) 15, nausea, muscle twitching; 11 nausea; 22 nausea and vertigo; 44 nausea.

(b) 13 convulsed; 19 muscle twitching; 41 nausea.

A remarkable feature was that seven of the twenty divers showed consistently high oxygen tolerance in four to six dives at 60 minutes, some finishing with a dive of 120 minutes. Although the Royal Navy had a few divers with almost consistently high performance we never had such a large proportion of highly resistant subjects. The difference in the two studies is discussed below. It was also reported by these authors that the degree of variation of individual tolerance was even greater at 80 and 100 feet, although no evidence is presented.

The wide variety of symptoms, even in the same diver, the minor crises, usually with nausea, the muscle twitching and convulsions with or without warning, were all encountered. In the analysis of the incidence of various symptoms, all symptoms in the wet (exercising) and in the dry (resting) have been combined. It is therefore difficult to make a comparison as it was found in the Royal Navy series that the more subtle symptoms (restlessness, excitability and other 'dysphorias') are far less frequent in a diving suit underwater, particularly with exercise. The high incidence of nausea and vertigo (57%) reported by Yarbrough is almost

certainly related to exercise underwater (see Chapter 2). The figures given for 'muscular twitching' is 21% (168 end-points). This figure was much higher in the Royal Navy underwater series both at rest and during exercise (60% approx. 508 end-points) and in almost all cases the twitching was in the lips. In the Royal Navy resting series in the wet there was occasional twitching of other parts (32 in 388 end points) but none was reported in working underwater exposures. Royal Navy oxygen divers considered lip twitching to be the cardinal warning symptom of an impending convulsion. It is possible that most of the Royal Navy experimental divers in World War II, of necessity, sailed a little closer to the wind as evidenced by a higher rate of convulsive end-points.

On the whole the oxygen tolerance of the subjects in Yarbrough's study appears to be greater than the tolerance of the subjects in the Royal Navy series. There are a number of factors which may have contributed to this apparent difference.

Dilution of Oxygen with Nitrogen: This is a considerable problem in exposures in compressed air. In the Royal Navy studies in the dry (1943), despite six preliminary vigorous purges of lungs and apparatus, washouts every 5 minutes for 30 minutes, and washouts every 10 minutes thereafter, nitrogen levels of up to 7% (Van Slyke volumetric method) were found in the respiratory circuit. This was, no doubt, due to mouthpiece leakages and considerable diffusion into the apparatus due to the high nitrogen gradient.

In Yarbrough's series in the dry he employed an open circuit with demand supply of oxygen and the FIO_2 would have been of the order of 0.95 and not dissimilar to that in Donald's dry series with multiple washouts and excess oxygen flow. This almost certainly explains the results in the two studies being compatible, with oxygen tolerance in the dry being of the same order. However in contrast to this state of affairs, there were critical differences in procedure in the Royal Navy and United States Navy studies underwater (wet chamber).

In the underwater experiments the British divers first purged six times and then breathed oxygen at or near atmospheric pressure for 8 minutes (inspection, testing for leaks) and for a further two minutes during compression. The dive was timed from arriving 'on bottom'. Oxygen flow into the apparatus was 1.2 1/min. in the resting dives (VO_2 0.3 1/min.) and 2.2 1/min. in the working dives (VO_2 1.0-1.4 1/min.). Thus in all the

British oxygen dives there was about 1 litre of excess oxygen rinsing the circuit every minute. The only further source of nitrogen underwater is that dissolved in the subject's body tissues and this was estimated to be less than half a litre on arrival at bottom. Almost all the divers occasionally by-passed their reducing valve and rinsed the counterlung with oxygen. This was a "psychological" exercise but was not discouraged unless excessive. Again the constant flow of oxygen was somewhat higher at pressure as these reducing valves (1942) were not pressure compensated. It was, and is, considered tolerably certain that the Royal Navy divers were breathing oxygen levels near to those of the cylinder gas (99%). As it will be seen later, Piantadosi et al (1979), using the same technique of careful purging and constant excess oxygen flow, achieved oxygen concentrations of 99%.

The United States Navy report (Yarbrough et al) does not describe any purging or washout procedure but it is thought that this was only done once before diving. In their paper, they stated that the average percentage of nitrogen in the closed circuit apparatus in 48 underwater dives at 40 fsw was 14% and in another series of 30 fsw (17 underwater exposures) it was 9%. This would not have occurred with adequate "lung rinsing". At 40 fsw, 14% nitrogen would reduce the true oxygen depth by 10 fsw and at 30 fsw, 9% would reduce it by 6 fsw.

Schaefer et al (1949) later measured the fraction of inspired oxygen (FIO_2) when breathing from the self contained closed circuit "rigs" (Browne & Lambertsen) which had been used in the Yarbrough (1947) study. Again no washout or purging drill was specified nor was an excess flow of oxygen used. It is probable that a single washout of variable vigour was carried out in both underwater studies (Yarbrough, 1947 and Schaefer, 1949). In Schaefer's study 50 divers swam at about 0.9 mph at 20 to 40 fsw for up to 1 hour. Four minute rests were taken every 15 minutes. The inspired oxygen fraction measured at the end of these 15 minute dives varied from 0.5 to 0.93 (average 0.79).

Thus it is tolerably certain that the fraction of inspired oxygen and the equivalent oxygen depth were considerably less in the 1947 United States Navy underwater studies than those in the Royal Navy (1942-45) studies (multiple washouts and excess oxygen flow. This would account for the apparent difference in underwater oxygen tolerance in the two navies which, at one time, became almost legendary, particularly as underwater

decompression stages as deep as 60 fsw were employed by the United States Navy up to the mid-1960s.

NOTE: Yarbrough et al (1947) carried out a smaller underwater series at 30 and 40 fsw using a demand valve and open circuit (FIO_2 0.95+). For the sake of clarity these open circuit oxygen dives have been excluded from the main account above, which involved only closed circuit dives. Not surprisingly the open circuit underwater oxygen divers (exercising) showed a moderate increase of toxic symptoms i.e. at 40 fsw 35% (8 of 23 subjects) had toxic symptoms in a two hour period compared with 23% (11 of 48 subjects) at the same depth in subjects with a closed circuit.

Pressure of Dive: The depth of dives recorded in the Royal Navy series was the pressure of air above the water. Thus the standing diver's upper chest was at 2 to 3 fsw greater pressure than the gauge reading. No note is made in the United States Navy report but, from other studies it is likely that the dive depths recorded were those at the diver's chest level. This again, would have contributed slightly to the apparent difference of oxygen tolerance in the two series, particularly between 30 and 50 fsw (see shape of oxygen tolerance curves in chapter 2)

A most interesting experiment described in this report by Yarbrough et al was one in which subjects breathed oxygen at pressure while standing submerged in water up to the neck. It was reported that the oxygen tolerance in this condition was similar to that "in the dry". No details are given. One tentative theory concerning increased toxicity underwater was that the cerebral circulation might be increased while standing underwater due to the hydrostatic effects.

The conclusions reached by the Royal Navy and the United States Navy after these large series of oxygen dives were very similar:

Donald (1945): "The variation of tolerance between individuals, the variation of tolerance of each individual, the impairment of tolerance with work and under water, all make diving on pure oxygen below 25 feet of sea water a hazardous gamble."

Yarbrough et al (1947): "For underwater work the safe inhalation of pure oxygen is limited to a depth of 30 feet."

There is no doubt that the introduction of these depth limits removed most of the dangers of oxygen diving. After World War II there was an enormous expansion of underwater fin-swimming in the armed forces, in commercial practise and in sports diving. The free venting of gas was no

longer a problem and ample gas supply with large cylinders allowed the use of demand open-circuit air diving.

However the need for closed circuit oxygen breathing during certain covert operations continued. In the Schaefer study (1949) mentioned briefly above, fin-swimming oxygen breathing divers were observed under controlled pressure conditions for the first time. The swim circle in the Submarine Escape Tank lock was about 36 feet and the rate of swimming about 0.9 mph, gauge pressures of 20, 30 and 40 fsw were employed. The water temperature was 90°F. Apart from the wide range of FIO_2 observed, there was a considerable frequency of symptoms reported suggesting oxygen poisoning, there being 14 instances in 50 one and a half hour dives. As FIO_2 was under frequent observation (during 4 minute rests every 15 minutes) the partial pressure of oxygen being breathed could be determined and expressed in fsw (equivalent oxygen depth; DO_2)

The 14 dives with symptoms were as follows. All dives terminated at the time indicated below.

Diver	DO_2 (fsw)	Time of dive (min)	Symptoms
A	34	30	Clonic Convulsion
B1	6	72	Nausea, fatigue
B2	11.5	40	Facial twitching, fatigue
B3	28	45	Nausea, vomiting, fatigue
B4	30	63	Facial twitching, fatigue
C1	20	72	Panting
C2	13	45	Facial twitching, dyspnoea
C3	29	88	Exhilaration, disorientation, Inspiratory inhibition
D	4.5	82	Clonic Convulsion
E1	22	42	Loss of co-ordination, fatigue
E2	30	55	Transient Unco', Clonic Movements L.Leg, Expiratory Inhibition
F	33	89	Panting
G	28	45	Dyspnoea
L	23	96	Nausea, facial pallor, inco-ordination

Thus at a DO_2 of 25 to 30 fsw many swimmers had symptoms of oxygen poisoning, one convulsing after 30 minutes at a DO_2 of 34 fsw and one 'near convulsing' (Transient unconsciousness and clonic movements) after 55 minutes at a DO_2 of 30 fsw.

However in the case of the swimmer convulsing after 82 minutes at a DO_2 of 4.5 fsw and of the two divers with facial twitching at a DO_2 of 11.5 and 13 fsw, errors in gas collection (glass tonometers and mercury) or analysis (Van Slyke volumetric) are most likely. The purported FIO_2 of the diver convulsing at a DO_2 of 4.5 fsw was reported to be as low as 0.513 after 82 minutes swimming.

Lanphier and Dwyer, USN (1954) felt that the limitation in the use of 100% oxygen in diving for 30 minutes only at 30 fsw was "not very useful" They wished, despite the Royal Navy and United States Navy findings, to dispense with this depth limit and if possible, to establish "safe" time limits at depths greater than 30 fsw. In their study they carried out 49 dives (19 subjects) at 5 foot stages from 20 to 45 feet. The oxygen was supplied by an open circuit ($FIO_2 > 0.95$). The work rate of these divers was "greater than a man could voluntarily sustain under diving conditions." To allow a very high rate of work, three types of exercise were practised in rotation, stationary swimming, weight lifting and underwater cycling. The water temperature was 80°F and the divers were "near to exhaustion" at the end of these dives. They were anxious to avoid severe toxic end-points and particularly convulsions. They therefore laid down "a rather arbitrary time-depth limit curve" from 20 to 45 fsw by "educated guessing" and from "previous field experience". This they called the "working limit curve". They added 25% to the time at each depth and called this the "test limit curve" (see Figure 19).

Dives at each depth were only continued to this "test limit" time:

20 fsw	No symptoms were encountered after 113 minutes. (10 subjects).
25 fsw	No symptoms were encountered after 81 minutes. (5 subjects).
30 fsw	No symptoms were encountered after 57 minutes. (11 subjects).
35 fsw	Out of 5 subjects, three reached the "test limit" time, (42 minutes) "safely", one had nausea at 28 minutes and one convulsed at 42 minutes.
40 fsw	Out of 13 subjects, 10 reached the "test limit" time (30 minutes), one had tinnitus at nine minutes and one vertigo at 17 minutes.
45 fsw	Of five subjects, 3 reached the "test limit time" (18 minutes), one had tinnitus at nine minutes and one vertigo at 17 minutes.

US NAVY OXYGEN DEPTH-TIME WORKING LIMIT (AFTER LANPHIER, 1954)

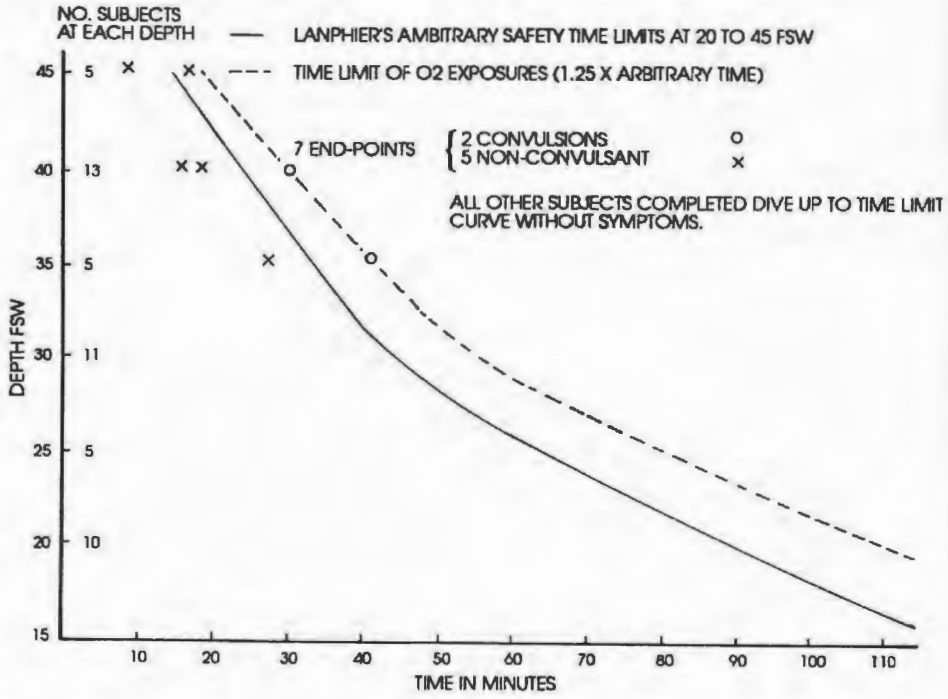

FIG. 19: Illustrating Lanphier's series of dives on pure oxygen between 20 and 45 fsw. The ambitiary time limits, the maximal exposure allowed and number of subjects at each depth are shown. Times of occurrence of symptoms causing cessation of exposure (end-point) are also plotted.

Lanphier (1955, (i)) noted that "no unequivocal symptoms (convulsions) occurred within the time limits of the proposed working limit curve." The "minor symptoms" of the other five subjects, four of which were within these times, were not "considered very convincing". The two convulsions and five non-convulsive end-points and the working limit curve are plotted in Figure 19.

After these investigations the so-called working limit curve (depth v time) was adopted. Thus the United States Navy standard oxygen diving limit curve was Lanphier's arbitrary working limit curve supported by the study just described.

Two other decisions were taken. The depth for oxygen diving for normal operations was reduced to 25 fsw as in the Royal Navy and time limits were also given at the shallower depths. Dives on oxygen deeper than 25

fsw were only allowed for "exceptional operations" (covert operations or emergencies) and the times were those of the United States Navy working limit curve, thus:

NORMAL OPERATIONS		EXCEPTIONAL OPERATIONS	
Depth fsw	**Time (min)**	**Depth (fsw)**	**Time (min)**
10	240	30	45
15	120	35	34
20	90	40	25
25	65	45	15

These limits were in use up to the mid-1970s (Wood, 1975). Despite the clear statements in the US Navy Diving Manual concerning the hazards of oxygen dives to depths greater than 25 fsw, the US Navy "standard" oxygen time limits up to 45 fsw for working dives have been illustrated and quoted in standard text books for many years without the strict reservations always being mentioned. This curve has also been used by biomathematicians in an unsuccessful attempt to develop a formula to estimate the risk of convulsions at any specific point during a series of oxygen exposures (Hill & Dossett, 1968).

Up to 1979 little more oxygen diving research was done in the US Navy apart from a study by Alexander and Flynn (1971) who exposed nine immersed working divers (swimming and weight lifting) to oxygen at 12 fsw for four hours without evidence of pulmonary or central nervous system toxicity. Water temperature was 85°F.

The Royal Navy continued to use the 25 fsw (actual 8m) limit for swimming oxygen divers but allowed the use of oxygen for 10 minutes at 33 fsw (10m) by booted divers doing light work (only up to 1987). No time limits were given for oxygen diving at or less than 25 fsw, but canister endurance and safe cylinder capacity drill usually limited exposures to 90 minutes or less. The Royal Navy did not specify time limits for "exceptional operations" at depths over 25 fsw. Each covert operation received individual consideration, including appropriate training. This long standing and, in my opinion, excellent practise has been changed recently (1988).

Further Exercise Studies in the Dry

In 1971 Young reported a large series of exposures of fully dressed and geared (60 lbs) fireman to pure oxygen in the dry at moderate pressures (about 2 ATA). They alternatively lifted heavy hose (VO_2 1.2 1/min.). They wore a PROTO oxygen breathing apparatus. Inspired gas PO_2 was determined and equivalent oxygen depth (DO_2) calculated. Although the relationship of some of the reported symptoms to oxygen poisoning remain in doubt, particularly very early prolonged lip fibrillation, the original protocols indicate that two subjects undoubtedly convulsed, one after 19 minutes at a DO_2 of 34 fsw (2 ATA) and one immediately after decompression to 10 fsw (dry) after 40 minutes work at a DO_2 of 38 fsw (2.15 ATA). It is possible that carbon dioxide levels in the breathing apparatus were considerably raised in some of these experiments. There was also a very marked degree of heat stress.

Prolonged Oxygen Exposure in Immersed, Exercising Divers at 25 & 30 fsw

Piantadosi, Clinton and Thalmann (1979): reported a most interesting and detailed study in which 6 divers exercised at 25 fsw underwater (wet chamber) for long periods while breathing oxygen.

The first series (6 subjects) was a study of canister performance in warm (21°C) and cold (4°) water. The divers were wearing Draeger's LAR v SCUBA with a 4.3 kg Sodasorb canister. The PCO_2 and PO_2 of the inspired gas was monitored throughout. "Careful" purging was carried out until an FIO_2 of 0.95 was obtained. Continuous excess oxygen flow (volume not specified) during the experiment "quickly increased" FIO_2 to 0.99. During the experiments the subjects pedalled on an ergometer at 50 W (VO_2 1.7 1/min. approx.) for 6 minutes followed by a 4 minutes rest (mean VO_2 of each 10 minute period 1.3 1/min. approx.). This sequence was continued until inspired PCO_2 reached 7.6 mm Hg, or for a maximum of 5 hours.

In the warm water dives the exposure time was 271 to 252 minutes. No run was stopped because of impaired canister performance (as defined). One diver discontinued when he reported tinnitus at 178 minutes and one at 180 minutes because of the accidental loss of cylinder oxygen. No other subject had symptoms of acute oxygen toxicity. However, of the 3 divers whose exposures were over 4 hours (249, 271 and 252 minutes), the latter two complained of mild "pleuritic" pain and retrosternal burning

associated with small changes in forced vital capacity and forced expiratory volume. These were the longest exercising oxygen dives ever reported at this depth (25 fsw) and the symptoms strongly suggested early pulmonary toxicity for the first time after an experimental working dive.

In the cold water canister series the time on oxygen was shorter (mean 163 ± 22 minutes) because canister effluent PCO_2 reached 7.6 mm Hg and the exposure was terminated. There was no symptom of oxygen poisoning or hypercarbia.

The second series (6 subjects) was of graded exercise of a severe degree. VO_2, peak end-tidal PCO_2, inspired PCO_2 and maximal respiratory pressure range at the mouthpiece were recorded throughout. Again, the whole study was performed in warm (21°C) and cold (4°C) water at 25 fsw. The following demanding procedure was carried out. Ten minutes at rest were followed by seven 10-minute cycles of 6 minutes work and 4 minutes rest. Exercise was increased in 25 W stages from 25 W to 150 W (VO_2 1.6 to 3.2 1/min.).

In the warm water the canister remained effective and inspired PCO_2 did not rise above 0.6 mm Hg at any degree of exertion. The peak end-tidal PCO_2 rose from 43 to 45 mm Hg (mean) at maximal exertion. No end-tidal PCO_2 over 48 mm Hg was recorded. Incidentally, the mean pressure swing (as a rough estimate of resistance) increased from 10 to 21 cm H_2O. It is noteworthy that, with standard oxygen breathing apparatus, there was no evidence of carbon dioxide retention despite 70 minutes of moderate (VO_2 1.6 1/min.) to very severe exercise (VO_2 3.2 1/min.).

In the cold water series the absorbent was not so efficient and the mean inspired PCO_2 rose from 0.6 to 3.2 mm Hg (rest to 150 W). There were no peak end-tidal PCO_2 values above 48 mm Hg up to and including the 125 W stage (VO_2 2.4 1/min.). At 150 W (VO_2 3.2 1/min.) the inspired PCO_2 was 2 to 4.5 mm Hg and only two out of six divers showed a rise of end-tidal PCO_2 above 48 mm Hg (54 and 51).

There were no symptoms of oxygen toxicity during the whole study (twelve 80 minute exposures). Finally, and most importantly, in all these long and very vigorous dives at 25 fsw on oxygen there was no evidence of carbon dioxide retention as judged by end-tidal PCO_2. When the canister performance was deliberately impaired by very cold water (4°C) and tested against heavy exertion there was again no important trend to hypercarbia (see Chapters 5 and 6).

In 1984 Schwartz carried out a similar series of canister performance studies during prolonged moderate and more severe graded exertion. The "rigs" being tested were the Emerson and the Fenzy PO8 oxygen breathing underwater apparatus. The protocol was almost identical to that in Piantadosi's 1978 study described above. Again FIO_2 was 0.98-0.99. The only important difference in this study was the greater depth (30 fsw) of the oxygen dives throughout.

In the 14 moderate exertion runs, aimed at 4 hours, three possible instances of oxygen poisoning occurred, as follows:

220 minutes; nausea and "visual disturbances"

152 minutes; vertigo and fatigue

165 minutes; lip twitching, dissociation and nausea

In the 17 graded exercise runs which were aimed at about 100 minutes (up to 150 W, VO_2 3.2 1/min.) two divers suffered from severe oxygen poisoning. The first, having reached the 150 W level of exertion, convulsed without warning at 90 minutes. He recovered after 18 minutes and did not suffer retrograde amnesia. Post-handgrip relaxation was impaired for a short period. The second diver experienced tunnel vision, dyspnoea and malaise at 83 minutes (100 W exercise level). He abandoned the dive, started climbing the ladder and switched to air breathing. He then appeared unable to move or speak for several minutes. He then continued to climb out of the "wet pot" with some help, spoke briefly to the medical officer and then lay down on a bunk, passing into a stuporose condition which lasted about two hours before he recovered his faculties. He had retrograde amnesia from the end of the dive to the time of awakening. At no time during or after the dive were there any local or generalized clonic movements. This most unusual syndrome had only been reported once previously. (Donald, 1945).

Inspired carbon dioxide tensions were monitored throughout this study and there was no significant or important rise of $PICO_2$ in any subject suffering from oxygen poisoning. The not infrequent occurrences of acute oxygen toxicity at a depth only 5 fsw deeper than in Piantadosi's study is noteworthy.

Further Studies at 40 fsw and 25 fsw with Intermissions

The next study in this series **(Butler & Thalmann, 1984)** was to examine afresh the standard US navy depth/time limits for closed-circuit working

oxygen diving (already described) with a view, if possible, to lengthening the then current exposure times. The current times they gave from the US Diving Manual (1973) were ascribed to Lanphier's 1954 study. They were however considerably different to his standard oxygen working limit figures being much shorter at depths below 30 fsw, i.e. 10 instead of 25 minutes at 40 fsw (see Limit II and Limit III in Figure 20).

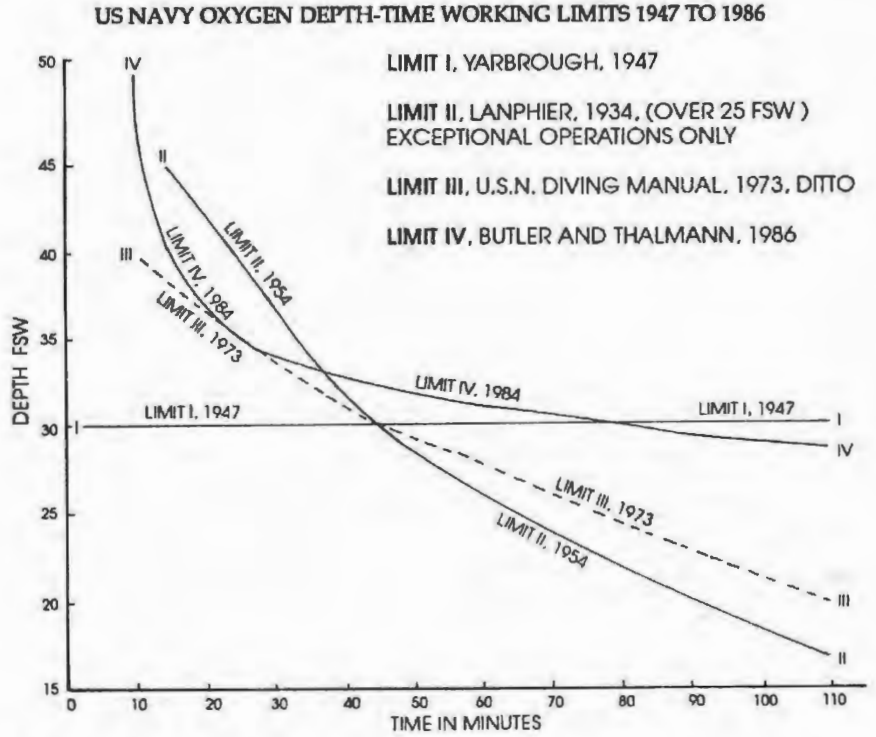

FIG. 20: Illustrating U.S. Navy oxygen depth-time working limits (with exercise). Note that limits II and III could only be used over 25 fsw in exceptional operational conditions. This does not apply to limit IV (Butler & Thalman, 1986).

The "safe" time at 40 fsw was first studied. Work was carried out on the underwater ergometer at 50 W (VO_2 1.7 1/min.). The time then recommended was 10 minutes. Lanphier's technique of limiting exposure to a "relatively unambitious" time was again used. 18 subjects were dived on oxygen to 40 fsw for a maximal time of 20 minutes. Two divers had "light headedness" (12 and 20 minutes) and 2 divers convulsed (20 and 19.5 minutes). 14 subjects had no symptoms. This dive was then repeated by 24 subjects with a maximum time of 15 minutes. There were no signs or

symptoms of oxygen poisoning and this figure (5 minutes more than the current standard depth/time limit) was recommended.

The second series in this study was to discover the effect of previous shallow oxygen exposure (25 fsw) on subsequent exposure to a greater depth (40 fsw). In the longer dives to 25 fsw the 6 minute work (50 W) 4 minute rest cycle was used. In the shorter intermissions at 40 fsw the divers worked continuously (50 W).

In order to assess the degree of safety in this series the authors felt that the following classification of symptoms would be useful:

(a) **Convulsions**
(b) **Definite:** included muscle twitching, tinnitus, blurred or tunnel vision, disorientation, aphasia, dysphasia, nystagmus or inco-ordination.
(c) **Probable:** more "equivocal" episodes that may have been caused by oxygen toxicity e.g. light headedness, apprehension, dysphasia, lethargy, transient nausea.

The divers condition, whether he stopped or not, and the pattern of symptoms in time were also taken into account before classification.

The following continuous profile was first tested: 60/25, 15/40, 60/25 (time in minutes/depth in fsw)).
13 exposures were carried out. Twelve subjects had no symptoms and one had blurred vision, light headedness and nystagmus, ("definite") after 14.5 minutes at 40 fsw.

The next profile was: 120/25, 15/40. There were fourteen dives. Eleven subjects completed without symptoms. One convulsed after 72 minutes at 25 fsw, two had "definite" symptoms in the 40 fsw phase of the dive, one after 8 minutes and one after 14 minutes. The authors concluded that brief excursions to 40 fsw were possible after prolonged exposure to oxygen at 25 fsw. They stated that "pre-exposure of 60 minutes at 25 fsw did not seem to influence the safe 40 fsw exposure time, but a 2 hour pre-exposure did seem to decrease the safe 40 fsw exposure somewhat."

A most unexpected finding was the oxygen convulsion after 72 minutes exposure at 25 fsw. A total of 63 working dives at 25 fsw had been conducted by the authors with exposures ranging from 81 to 252 minutes with only one subject complaining of "ringing in his ears" after 178 minutes at 25 fsw. The authors state: "based on these results, our expectation was

that the exposures at 25 fsw would result in less serious oxygen toxicity symptoms, but not produce any convulsions." The present author would not accept the inference that time/depth exposures or even depth exposures can be conveniently divided into those where "less serious" oxygen toxicity symptoms, without convulsions, might occur and those where convulsions occur.

Butler and Thalmann (1986)

In the next series they continued to attempt to define the "safe limits" breathing oxygen at 25 to 50 fsw. All dives were working dives as described above (VO_2 1.7 1/min.).

25 fsw: In view of the convulsion after 72 minutes at 25 fsw (see above), 22 exposures were carried out for 4 hours. There were no symptoms.

30 fsw: The first 37 exposures for 90 minutes at this depth caused no symptoms. The next diver felt nauseated after 76 minutes and convulsed at 82 minutes. Three more dives to 82 minutes were symptomless. A safety time of 80 minutes was recommended.

35 fsw: There were 40 exposures aimed at 30 minutes. 35 subjects had no symptoms. One subject had "probable" symptoms (hearing impaired, tingling all over) at 12 minutes. Another four subjects had "definite" symptoms between 25 and 29.5 minutes (muscle twitching, aphasia, tinnitus, impairment of hearing etc.). 47 dives for 25 minutes were then carried out. There were no symptoms suggesting oxygen toxicity and so a 25 minute limit at 35 fsw was recommended.

40 fsw: It will be recalled that after two convulsions (at 19.5 and 20 minutes) in a group of 18 subjects, the time was reduced to 15 minutes and there were no symptoms in 24 exposures. Forty more exposures for 15 minutes were carried out without symptoms, and this time was recommended as the limit at this depth.

50 fsw: There were no symptoms in 57 exposures for 5 minutes. In 58 exposures for 10 minutes, only one subject complained of symptoms (tingling and vertigo at 9 minutes ("possible")). Ten minutes was recommended as the limit at this depth.

The following working dive oxygen exposure limits were recommended and are now official:

Depth (fsw)	Time (minutes)	Depth (fsw)	Time (minutes)
20	240	35	25
25	240	40	15
30	80	50	10

Let us briefly recapitulate the previous data in relation to these recommended times:

25 fsw: There had been a convulsion after 72 minutes at this depth, on oxygen, below the safety time (240 minutes).

30 fsw: There had been a convulsion after 82 minutes at this depth and yet the "safety" time is 80 minutes.

35 fsw: A subject suffered diffuse twitching of the leg muscles after 25 minutes and had to discontinue the dive which was aimed at 30 minutes. Three other divers had "definite" symptoms between 25 and 29.5 minutes while attempting 30 minutes at this depth.

40 fsw: Divers convulsed at 19 and 20 minutes at this depth, yet the safety time limit is only 4 and 5 minutes less i.e. 15 minutes.

50 fsw: One subject had parasthesiae and vertigo at 9 minutes.

It will be recalled (Chapter 2) that in the Royal Navy studies (1945) there were a considerable number of instances of oxygen poisoning at 25 to 50 fsw during working dives in times well below the proposed safety limits. Generalised convulsions occurred at 30 fsw in 10 minutes (working) and in 42 and 48 minutes (non-working). Convulsions also occurred in working dives at 6 and 10 minutes at 50 fsw.

The new "safety" times recommended are, in most instances, only a few minutes less than times causing convulsions and other forms of acute oxygen poisoning at that depth in their own studies. Considering that we are dealing with biological phenomena, this is a somewhat unreal precision exercise. It is impossible to reconcile these recommendations with the extreme variation of tolerance in individuals and between individuals so clearly shown by Donald (1945) and Yarbrough et al (1947). It is also possible that, on occasions, some oxygen sensitive subjects were eliminated in the first exploratory series of dives at each depth. There is a touch of "last one across the road", with the casualties depending on the unknown factors determining oxygen tolerance of particular individuals on a particular day. These recommendations are for the indefinite future for new groups and new individuals. The risks due to variability are not so great at the lower end of the tolerance distribution in large groups, but unpredictable danger is there all the same.

The authors discussed the problems in "establishing limits". They stated: "clear-cut groupings of toxicity episodes . . . require no agonising and establishing the safe exposure limit is straight forward." They continue: "more difficult decisions must be made when faced with single toxicity episodes." They mention "the need to strike a workable balance between single oxygen toxicity episodes (these include convulsions) and operational safety." They acknowledge "the possibility of occasional episodes of oxygen toxicity."

Yet they state later in the same article "exposure limits below 25 fsw are felt to be well tested and should be considered safe enough for routine operational use," (whatever operational means). In introducing these limits for use in the US Navy Diving Manual (1985) they stated *"these new limits have been tested over the entire depth range and are acceptable for routine diving operations. They are not considered exceptional exposures."* The present author would strongly oppose the acceptance of the possibility of acute oxygen poisoning in the oxygen exposure time limits recommended for routine diving operations. Such an acceptance could impair the traditional and essential trust between divers and those responsible for their safety.

The rest of the article (Butler and Thalmann, 1986) concerns the effect of long (2 and 4 hour) pre-exposures at 20 fsw on the oxygen tolerance during intermissions at greater depths (i.e. 25/35). This part of the investigation obviously relates to covert operational work, although it is published "in the open". Their conclusion were as follows:

1. A pre-exposures depth of 20 fsw has much less effect on a subsequent excursion than 25 fsw.
2. 20 fsw pre-exposures for 2 and 4 hours seem to slightly increase the possibility of an oxygen toxicity episode on subsequent excursions to 35 fsw but there is no consistent difference between the effects of these two pre-exposure periods.
3. A return to 20 fsw for periods of 95 to 100 minutes provided an adequate recovery period from an earlier excursion (25 minutes at 35 fsw) and enabled a second excursion (25 minutes at 35 fsw) to be taken without additional hazard.

For good reasons, these conclusions were considered somewhat tentative. The suggestion that a subject could recover from incipient oxygen poisoning

caused by an initial deeper excursion when he returned to 20 fsw is interesting but certainly not proven. The authors themselves mention the possibility that the first deeper excursion may have eliminated the most oxygen sensitive subjects in the group and that this led to better performances in the second deeper excursion.

The final paper of this series (Butler, 1986) will not be reviewed in any detail. It is, again, mainly concerned with covert approaches to military targets. All are working oxygen dives. During prolonged exposures to 20 fsw, two or even three excursions to a greater depth (15 minutes at 40 fsw) were carried out. The intervals at 20 fsw between excursions were increased from 30 to 90 minutes. The results were not encouraging as a significant number of divers suffered from acute oxygen poisoning during an excursion. 19 out of 43 subjects had minor symptoms of pulmonary oxygen toxicity after a 4 hour '20 to 40' fsw oxygen profile, complaining of cough and sub-sternal discomfort. These divers had two or three excursions of 15 minutes at 40 fsw during the 4 hour period.

However, a most striking and important finding was the unexpected occurrence of acute oxygen poisoning in 4 out of 11 subjects after an initial 15 minute period at 40 fsw, followed by a proposed 90 minutes at 20 fsw before the second excursion to 40 fsw. The whole dive programme was 15/40, 90/20, 15/40, 90/20, 15/40, 15/20. During the first stay at 20 fsw the following episodes occurred:

Subject	Time (min) At 20 fsw After Initial 15/40 Exposure	Symptoms & signs	Authors' Syptom Classification
A	31	Nausea, vomiting, facial paraesthesiae. Dive terminated	Probable
B	48	Muscle twitching, dyspnoea, vertigo, before major convulsive attack.	Convulsion
C	68	Blurred vision, dyspnoea, vertigo, confusion. Dive terminated.	Definite
D	80	Nausea, severe tinnitus, arms went rigid in extension, non-responsive to signals to stop. Pulled off ergometer.	Definite

The remaining seven subjects completed the whole profile (15/40, 90/20, 15/40, 15/20) with only one subject having symptoms in the last 40 fsw excursion (severe apprehension, circumoral paraesthesiae, "possible").

The four serious episodes of oxygen poisoning at 20 fsw give us some new and extremely valuable information. The most likely series of events is that these divers were being intoxicated at 40 fsw but not to a degree causing overt symptoms. It is most unlikely that the stay at 20 fsw allowed reduction or reversal of the intoxication as these divers only developed acute symptoms of oxygen poisoning after considerable periods at this depth (31 to 80 minutes). A further degree of intoxication while at 20 fsw is the only feasible explanation. In previous dives; there had been a very occasional "probable" symptom of oxygen poisoning at 20 fsw (nausea, vertigo). More dives of this nature with long exposure to 25 to 10 fsw after exposure to say, 15 to 20 minutes at 40 fsw or 10 or more minutes at 50 fsw, would give new and important evidence of the possible sub-clinical neurotoxicity of oxygen at these shallow depths. Divers are told that they are "safe" for up to 4 hours at 20 to 25 fsw. *Yet here is strong evidence that neurotoxic events are occurring in the central nervous system while breathing oxygen at these depths.*

Oxygen Tolerance Test

In this study Butler tested three divers who were the most susceptible to oxygen toxicity in the whole series. All the divers, including these three, were highly trained and had undergone and passed the standard US Navy oxygen tolerance test (OTT). In this test the subject breathes oxygen while seated for 30 minutes at 60 fsw (2.82 ATA) in the dry pressure chamber. Further OTTs on these three "sensitive"subjects (10 tests on the first, 5 tests on the second and 2 on the third) were all completely negative. Most subjects are able to tolerate oxygen at this depth (dry) for 2 to 3 hours.

Butler and Knafelc (1986), in discussing the OTT, reported that, in the period 1972 to 1981, only 26 subjects had suffered toxicity episodes and that 10 of these had convulsed. The total number tested was 1347, giving a 1.9% incidence of "positive" tests. Nevertheless, it is concluded by Butler and Knafelc that although divers who have passed the OTT are able to tolerate the oxygen exposures routinely encountered in operational diving, this probably has little relation to their passing the undemanding OTT. They state that other navies consider the intra-individual variation so marked that they have discontinued the administration of screening OTT tests.

Butler suggests using a more severe OTT now that increased times of exposure to oxygen at 30, 40, and 50 fsw are allowed in the US Navy (see above). He does not consider this feasible without "the identification of a biological parameter of oxygen poisoning before overt symptoms occur." However, if one were seeking a more selective and severe OTT, Behnke (1942, (ii)) reported as early as 1942, although without giving data, that very moderate exercise at 3 ATA in the dry caused acute toxic symptoms in most subjects in about 20 minutes (see also Hayter and White, 1946). Behnke recommended "this exercise test as valuable in determining the oxygen tolerance of an individual." The ambient pressure and degree of exertion could be adjusted to obtain a meaningful "cut-off". Yet in over 40 years no effort has been made to adopt Behnke's proposal and little work has been done to explore further the remarkable effect of exercise in the dry during oxygen exposure. There must be a conscious or unconscious reason for this. Is it not possible that the formal OTT, although it excludes a small number of extremely susceptible trainees, is, of necessity, a ritual that reassures 98% of those tested that breathing oxygen within the official time and depth limits is reasonably safe? If the OTT were made more severe, a perfectly simple thing to do, then problems of individual and group morale could arise.

General Policy

Finally, it is worth considering what policy should be adopted with regard to diving on pure oxygen. To avoid confusion, let us first exclude covert military diving from our considerations. The ideal, in the author's view, would be a total ban on diving when breathing "pure" oxygen. The U.K. Services respond to this proposal by protesting that, in any case, they do not use "pure" oxygen for any routine diving purposes. Yet they show considerable reluctance to forbid routine oxygen diving outright. The Royal Navy Diving Manual states "Diving using pure oxygen is confined to initial training." The limit is 7m. Thus we have a strange situation where the gas used routinely to introduce trainees to self-contained counterlung diving is strictly forbidden when "initial training" is completed.

Although service regulations and the reputable diving clubs' rules could effectively forbid routine oxygen diving at any depth there are a

large number of independent self-contained divers who are not accessible to such control. Reports suggest that the use of oxygen by such divers is increasing. The wide dissemination of the recent US Navy oxygen working safety limits for routine diving down to 50 fsw, might lead to some independent oxygen divers re-entering depth zones that have been forbidden for forty years.

In view of all these difficulties, a simple and perhaps more effective exercise would be *to return to the world-wide position of 35 years ago when oxygen diving was universally limited to an agreed depth (25 fsw). 15 fsw would now be an appropriate limit.*

With regard to covert operations it would be far better for general diver-safety if no public mention were made of the higher tensions of oxygen or prolonged periods of oxygen breathing which may be hazarded by special service divers in such operations. There is also a dangerous tendency to consider that well trained and experienced divers, such as these, are less prone to acute oxygen poisoning. They may more readily appreciate premonitory symptoms, if they are fortunate enough to have them, but they are as vulnerable to oxygen poisoning as the youngest trainee on his first oxygen dive.

Diving clubs avoid the dangers of oxygen poisoning by the simple expedient of forbidding the use of any rebreathing apparatus by their members. Air breathing on demand in open circuit is universally used with a high degree of safety. There are strict depth limits, some absolute (usually 50m) and some related to competence and experience. Nevertheless most club regulations do not cover the possible use of oxygen-nitrogen mixtures in what are usually air-on-demand open circuit apparatus. Using such an apparatus, an oxygen-nitrogen mixture with an oxygen percentage as low as 40% would result in the partial pressure of oxygen in the inspired gas at the usual maximal depth (50m) being 2.4 bars (see Chapter 4). This practise is obviously dangerous in small groups of amateur divers and in the case of a single diver, it could be near-suicidal. It would be wise to prohibit the use by amateur divers of hyperoxic gas mixtures in either open or semi-closed breathing apparatus. (For further consideration of this important problem see chapter 5.)

REFERENCES

ALEXANDER, J.M. and FLYNN, E.T. (1971): Report of nine four-hour exposures to 100% oxygen at 11-13 fsw. *US Naval Experimental Diving Unit Research Report.*, 9/71.

BEHNKE, A.R. (1946): Personal communication to K.W. Donald. Reference, USN Forces, Europe, *Medical Liaison and Research.* A/11/E242/507

BUTLER, F.K. (1986): C.N.S. Oxygen toxicity in SCUBA Divers III, *United States Navy Experimental Diving Unit Report*, 5/86.

BUTLER, F.K. and KNAFELC, M.F. (1986): Screening for oxygen intolerance in US Navy divers. *Undersea Biomedical Research*, 13, 91-98.

BUTLER, F.K. and THALMANN, E.D. (1984): C.N.S. Oxygen toxicity in closed circuit SCUBA divers I., *Proc. 8th Symposium Underwater Physiology*, 15-30.

BUTLER, F.K. and THALMANN, E.D. (1986): C.N.S. Oxygen toxicity in SCUBA Divers II, *Undersea Biomedical Research*, 13, 193-223.

HAYTER, R. and WHITE, W.A. (1946): Oxygen poisoning in man, effect of exercise on time of onset of symptoms. *Naval Medical Research Institute, Bethesda, Report 2, Res. Proj.* X-436.

HILLS, B.A. and DOSSETT, A.N. (1968): Predicting the occurrence of oxygen convulsions. *Report 3/68 Royal Naval Physiological Laboratory, Alverstoke.*

LANPHIER, E.H. (1955, (i)): The use of nitrogen-oxygen mixtures in diving. *Proc. 1st Symposium Underwater Physiol., Pubn.* 377, Nat. Ac. Sc. & Nat. Res. Council, Wash. D.C., 74-78.

LANPHIER, E.H. and DWYER, J.V. (1954): Diving with self-contained underwater breathing apparatus (oxygen tolerance). *US Navy Experimental Diving Unit Report*, 8/54.,

PIANTADOSI, C.A., CLINTON, R.L. and THALMANN, E.D. (1979): Prolonged oxygen exposures in immersed exercising divers at 25 fsw. *Undersea Biomedical Research.*, 6, 347-356.

SCHAEFER, K.E., ALVIS, H.J., WEBSTER, A.P., WILLMON, T.L. (1949): Studies of oxygen toxicity, *U.S. Naval Submarine Medical Research Lab. Technical Report No. 149, New London.*

SCHWARTZ, H.J.C. (1984): Manned testing of two closed circuit oxygen U.B.A. *US Navy Experimental Diving Unit Report.*, 13/84.

WOOD, J.D. (1975): Oxygen Toxicity. *The Physiology and Medicine of Diving. Ed. Bennett and Elliot, 2nd Edition, Baillière Tindall,* London.

YARBROUGH, O.D., WELHAM, W., BRINTON, E.S. and BEHNKE, A.R. (1947): Symptoms of oxygen poisoning and limits of tolerance at rest and at work. *Experimental Diving Unit (USN) Report 1, Project X-337,* (Sub.No.62).

YOUNG, J.M. (1971): Acute oxygen toxicity in working man. *Underwater Physiol. Proc. of Fourth Symposium. Academic Press, N.Y. and London.*, 67-76.

CHAPTER 4

FURTHER CONSIDERATIONS OF OXYGEN POISONING. PULMONARY OXYGEN TOXICITY. UNDERWATER DECOMPRESSION BREATHING OXYGEN.

Pulmonary Toxicity

The toxic action of oxygen on the lungs (Lorrain Smith effect) is the lesser danger in oxygen poisoning. It does not have the dramatic and life threatening features of acute oxygen poisoning of the central nervous system. The rate of development of pulmonary toxicity is roughly proportional to the tension of oxygen breathed. It is mainly encountered during long exposures at low or moderate pressures when acute oxygen poisoning is less likely to intervene. A rough order of oxygen exposures to cause significant pulmonary damage and symptoms in most subjects is 18 hours at 1 AT, 7 hours at 2 ATA and 4 hours at 3 ATA.

The symptoms of pulmonary oxygen toxicity, in contrast to acute CNS poisoning, are remarkably consistent. Early pulmonary toxicity may only cause slight retrosternal discomfort. When fully developed, the symptoms resemble those at the onset of a virulent upper respiratory tract infection with painful awareness of the tracheo-bronchial tree. There is an irritative, often painful and sometimes uncontrollable non-productive cough. Both pain and coughing are markedly aggravated by a deep inspiration or by increased ventilation with exertion. Dyspnoea occurs even at rest.

It is tolerably certain that the breathing of oxygen tensions of up to 0.5 AT for "multi-day periods" causes no functional or structural pulmonary damage. Helvey et al (1962) exposed normal subjects to 0.49 AT of oxygen for 14 days without any apparent untoward effect. The maximal non-toxic level of oxygen is most important in saturation diving with long exposures.

Divers are "stored" at increased pressures. The breathing of air while "in storage" at 60 fsw gives a partial pressure of oxygen of 0.59 AT. Although it is probable that this partial pressure of oxygen has no deleterious effect, most operators prefer not to exceed 0.5 AT and many others are even more cautious and work at considerably lower levels (0.30 to 0.35) as these divers usually carry out a number of deeper excursions and may breath pure oxygen at some stages of decompression. It is also advisable to leave some margin for inadvertent gas composition variations.

The possibility of pulmonary irritation and damage in divers did not arise until the second World War when oxygen diving was carried out on an extensive scale for the first time. As reported by the author (1945), there was no clinical, spirometric (vital capacity) or radiological evidence of any pulmonary toxicity in any operational (including training) or experimental diving in large groups over a period of several years. In 1945 the author wrote, perhaps a little unwisely, "it can be stated that no real underwater dive will ever be made where lung damage will result from high tensions of oxygen." Nevertheless there were some unusually prolonged oxygen dives at "non-toxic" (CNS) depths, with deeper intermissions, during covert approaches to enemy ships. Pulmonary damage without the intervention of acute convulsive symptoms was a distinct possibility. A number of experimental dives were therefore carried out to explore this hazard. An example of such dives was (time in mins/depth fsw) 249/12, 30/50, 90/12. There was no clinical or spirometric evidence of any pulmonary effects in any such dives (see Chapter 2).

It was a number of years before more precise and systematic attempts were made to measure the risks and degrees of pulmonary intoxication by hyperbaric oxygen. The increasing use of therapeutic hyperbaric oxygen in a whole variety of diseases and clinical procedures also stimulated interest. Groups of subjects were studied breathing oxygen in the dry, at rest, at pressures between 0.8 and 2.0 ATA and the times of exposure causing a given percentage decrement (2 to 20%) of vital capacity were determined (Clark and Lambertsen, 1971; Caldwell et al, 1966 and Ohlsson, 1947). These studies showed a hyperbolic relationship between pressure and time causing a given degree (in 50% of the group) of pulmonary toxicity as judged by vital capacity changes (see Figure 21). The upper ranges of these pulmonary toxicity curves are obtained by extrapolation as acute oxygen

poisoning (CNS) will occur before even 2% decrement of vital capacity (DVC) is reached.

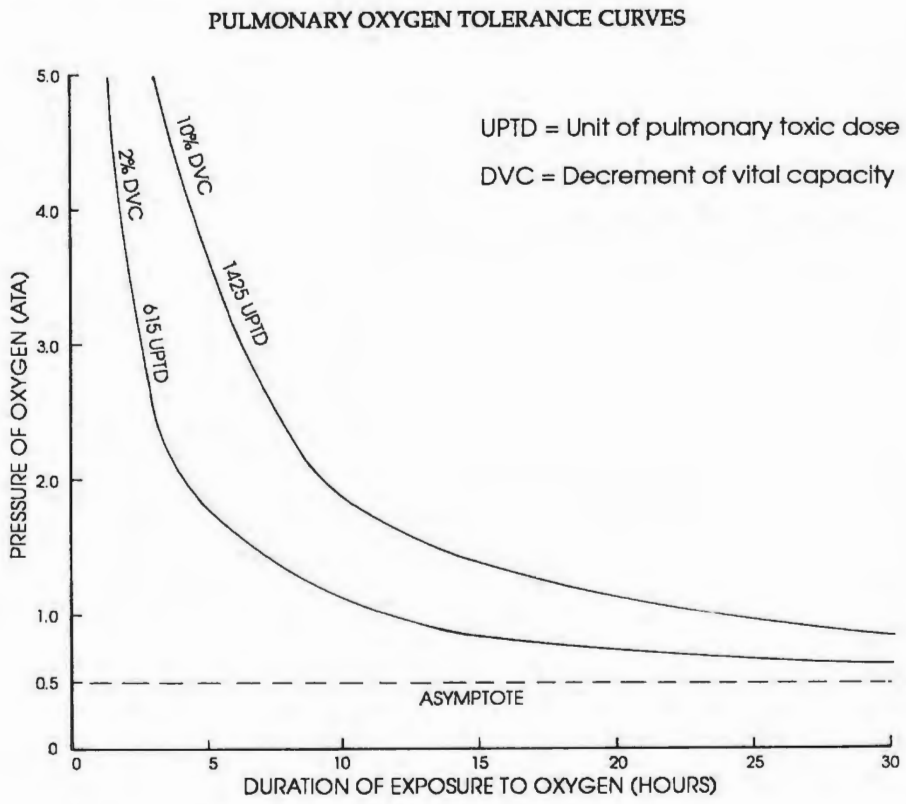

FIG. 21: Hyperbolia curves showing time of exposure to oxygen to cause 2 and 10% decrement of vital capacity (50% of group) at different pressure. Normal male subjects (after Clark & Lambertson, 1971).

A hyperbolic relationship between the time of onset of acute oxygen poisoning and the ambient pressure had first been reported in man by Donald (1945) in two separate studies on groups of oxygen divers at 50, 60, 70, 80, 90 and 100 fsw. In the first group (n = 5) the best fitting mathematical curve to the median values of these divers' times to end-point at different depths was a rectangular hyperbola as illustrated in Figure 9 (chapter 2) and representing the equation:

$$t = \frac{1086}{D - 39.7}$$ t in min, D in fsw

In the second study (n = 14) the relationship of the median values of divers' times to end-point to depth was represented by the equation:

$$t = 3.18\ D^{-3.82}$$ t in min, D in ATA

This relationship was, of course, linear when log.-log. co-ordinates were used (see figure 13, chapter 2).

It was also demonstrated in this study (Donald, 1945) that if the "dosage" of oxygen was simply measured in time – pressure units (time in minutes X pressure (ATA – O.21 AT), a "unit" dose of oxygen became more toxic at increased pressure.

Fixed percentages of the group (75, 50 & 25%) were "eliminated" by acute symptoms at 100 fsw (4 ATA) by about one third of the time-pressure dosage required at 50 fsw (2.5 ATA).

Bardin & Lambertson (1970) and Wright (1972) dealt with this problem of the changing toxicity of oxygen at different pressures by a most ingenious device. First they defined a unit of the pulmonary toxic dose (UPTD) as the degree of pulmonary toxicity incurred by breathing 100% oxygen at a pressure of 1 AT for one minute. They then calculated corrections to allow for changes of toxicity at all other pressures. This correction Kp

$$Kp = \sqrt[-1.2]{\frac{0.5}{p - 0.5}}$$

although derived mathematically, was, however, essentially describing the behaviour of the empirically determined hyperbolic pulmonary oxygen tolerance curves which incorporated changes in toxicity with changes in pressure. Thus at, say, 3 ATA the "dosage" is not 3 x exposure time (mins) UPTD but 3.82 (Kp) x time (mins) UPTD. Tables giving Kp at all pressures from 0.5 to 5.0 ATA or of UPTD "dosage" at different times at particular pressures are available (Wright, 1972). It is thus possible to state the degree of pulmonary oxygen toxicity suffered by subjects breathing oxygen in UPTD units at any time at any working pressure.

Subjects suffering at 2% reduction in vital capacity (50% of the group) had a dosage of 615 UPTD. This was considered a reasonable limit in ordinary diving and in routine hyperbaric exposures in the dry. Dosages of 1425 UPTD or over (10% or more DVC) are only considered justifiable

during the treatment of aero-embolism, grave decompression sickness or other mortal diseases (i.e. gas gangrene).

The initiators of this system of measuring pulmonary damage were well aware that, although useful general safety levels were now available, considerable caution was needed in the interpretation of an individual exposure. As with acute oxygen poisoning (Donald, 1945) there is a marked individual variation of pulmonary susceptibility. Thus there is a distinct possibility of greater pulmonary damage to sensitive subjects at, or even below, the average acceptable UPTD dose levels (614). Further there is no precise relationship between the dose in units of pulmonary toxicity, the DVC or the severity of symptoms in a single instance (Clark and Lambertson, 1971b).

In the studies of acute (CNS) oxygen poisoning in Chapter 2 the full range of oxygen sensitivity of all individuals in each group was determined under various conditions i.e. different depths in compressed air and under water, during rest and exercise. When recommending times of safety, the time chosen was near to or below the lowest ranges of oxygen tolerance found in each group. Although pulmonary oxygen toxicity is more insidious and less dramatic, it is surely prudent and proper to use the same safety criteria.

Clark (1970) and Clark and Lambertson (1971b) illustrated the variation of individual pulmonary tolerance, as judged by the time to cause 4% DVC in 10, 30, 50, 70 and 90% of a group of normal subjects when breathing oxygen at various pressures, with a series of different hyperbolic curves. If the same results are replotted as the time of exposure v. the percentage of the group suffering 4% DVC at a particular pressure, say 2 ATA, a typical Galton-Macalister skew distribution curve is found (see Figure 22), as was frequently demonstrated in studies of CNS oxygen toxicity (Donald, 1944, 1945; see Figs 1 and 12).

In Figure 22 the 90%, 50% and 10% UPTD "doses" to achieve 4% DVC during 3 and 2 ATA oxygen exposures are shown against the tolerance curves. The figures in parenthesis are the same "doses" restated in relation to the average required dose expressed as unity. It will be seen that at 2 ATA the "dosages" of oxygen causing a uniform degree of pulmonary oxygen poisoning, as judged by 4% DVC, vary from 660 to 1617 UPTD (10 to 90% of subjects). If all the subjects in this study at 2 ATA had been given the "dose" causing 4% DVC in 50% of the group (832 UPTD in 333 minutes), the

most sensitive diver would have been exposed for another 84 minutes after achieving his personal toxic "dose" (660 UPTD) with nearly 50% greater pulmonary damage than would obtain in the subject with average tolerance. Recovery in such a diver would obviously take longer. It will also be seen that the range of sensitivity, as judged by time or UPTD to attain 4% DVC, is of the same order at 3 and 2 ATA (1.9 to 0.75 of average). It is tolerably certain that similar ranges of sensitivity will be found in groups exposed up to a maximum DVC of 2%. Thus exposure levels as low as 460 UPTD per dive in the sensitive diver will cause a degree of pulmonary toxicity similar to that caused by the 615 UPTD average exposure. It is possible, therefore, that sensitive divers, who repeat exposures of 615 UPTD on several days may be more likely to suffer further lung injury before the trauma of the previous dive has fully healed.

VARIATION OF PULMONARY OXYGEN TOLERANCE

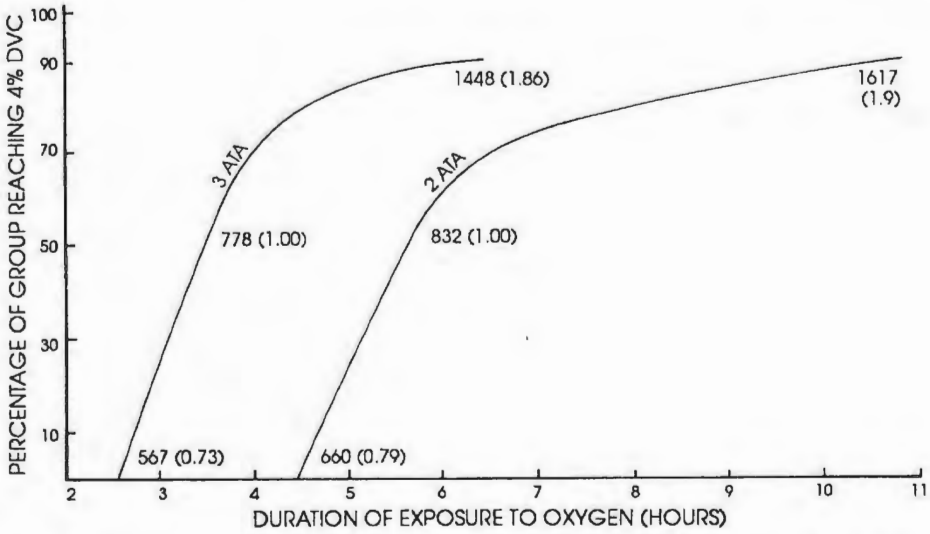

FIG. 22: Curves showing distribution of pulmonary oxygen tolerance in a group of normal men at 2 and 3 ATA as measured by time to achieve 4% decrement of vital capacity (DVC). Dosage UPTD to achieve 4% DVC shown for 10%, 50% & 90% of the group at each pressure. Ranges in parenthesis. Average dose as unity. Data approximate, derived from figure only (Clark & Lambertson, 1971a)

The above figures are only approximate but they strongly suggest that, in the case of divers carrying out prolonged or repeated dives involving some degree of hyperoxia, the sensitivity to pulmonary oxygen poisoning of

these periods may be divided, with up to two hours surface intervals on air between dives but this is certainly not the case in a number of tasks. It is now apparent that these periods are too long as is evidenced by the series of dives (Piantadosi et al, 1979 and Butler, 1986), described above. Another important consideration is the real possibility, discussed at some length in Chapter 3, that oxygen may have some fairly immediate neurotoxic effects at 20 to 25 fsw (Butler, 1986). If one must dive on oxygen, the total exposure allowed in 24 hours up to 25 fsw should be considerably less than 4 to 6 hours and more of the order of 2 hours. (UPTD 260), particularly if oxygen diving is being practised regularly.

Cumulative Damage

Many of the above considerations raise the question of possible cumulative damage in oxygen or hyperoxic diving. It had been concluded (Donald, 1945) that any such cumulative effect was most unlikely. Divers who were frequently exposed to raised and often toxic tensions of oxygen over three years had remained fit and active and had shown no apparent loss of special skills or of general competence. Clinical, spirometric and radiological studies showed no evidence of any pulmonary damage. However, in retrospect, these exposures were irregularly grouped in time and the majority were relatively brief with acute end-points (CNS). Again at that time (1942 to 45) the possibility of pulmonary toxicity occurring without notable symptoms or definite (10%) changes in vital capacity or in the x-ray appearance was not fully appreciated.

Sterk and Schrier (1985) and Sterk (1986) have again raised the possibility of cumulative (chronic) oxygen poisoning in accepted diving procedures. They consider that air divers carrying out long exposures of the order of 400 to 500 UPTD each day, sometimes on seven occasions in two weeks, are at risk, particularly if recompression with oxygen therapy is needed. They give examples of two such divers who had cumulative exposures of 3439 and 2568 UPTD over 12 and 13 days respectively. The former suffered a bend and had further hyperbaric oxygen during treatment. Both divers had tingling and numbness of the hands after these exposures, in one case for about eight hours. These authors consider that these paraesthesiae are an important symptom of chronic oxygen poisoning although such symptoms are relatively rare in very large series of acute

oxygen poisoning (Donald, 1945). The present evidence is certainly inconclusive.

The discussion earlier in this chapter concerning "sensitive" divers, with possible repeated damage before full recovery, may be relevant to this problem. Over-ventilation, with or without pulmonary damage, causing paraesthesiae is an attractive but unlikely hypothesis. Sterk regrets that there is no objective test of pulmonary toxicity, other than calculating UPTD dosage, available in operational conditions. It would certainly not be easy to measure the vital capacity in the field but the use of a simple peak flow meter (inspiratory and expiratory) might be worth investigating. There was some scepticism when the present author first suggested "it is possible that a simple whistle-like instrument to measure the maximum expiratory velocity will become a standard clinical tool" (Donald, 1953). Such an instrument was successfully developed by Wright (1958) and it is now universally used in primary clinical care. Perhaps, the same may happen in the diving world.

The nature and degree of change in the lung, CNS or any other organ in separate "safe" (sub-clinical) oxygen exposures and the rate of recovery from such changes, if they have occurred, is at present completely unknown. We must therefore await further evidence. The world-wide use of diving procedures of the type described above for many years without the manifestation of any objective or disabling disturbance makes chronic oxygen poisoning an unlikely possibility. Nevertheless there is a niggling doubt. It is very likely, as Bert proposed, that there is some abnormal activity, particularly at cell enzyme level, in many organs at all oxygen tensions above those encountered during evolution and in everyday life. The proposition that sub-clinical damage may be occurring and, on occasion, accumulating in divers must continue to be entertained. Meanwhile a considerable contribution to the whole problem will be to measure and know the pulmonary oxygen sensitivity of all divers who are regularly exposed to hyperbaric oxygen for significant periods.

The Use of Oxygen in Underwater Decompression

Oxygen was used to accelerate the elimination of nitrogen after air diving as early as 1917. This was during the recovery of the gold from the sunken *Laurentic* when, for many operational reasons (enemy action, weather

conditions), the time spent decompressing underwater had to be the bare minimum. The oxygen was administered immediately after surfacing from decompression in the sea. (Damant, 1951).

In 1930 the Royal Navy carried out a series of trials with the object of extending air diving to 325 fsw (Second Admiralty Deep Diving Committee). In these trials Damant shortened decompression time by the breathing of oxygen from 60 fsw upwards. These tables, known in the Royal Navy as "Damant's tables" were formally adopted in 1932 (Royal Navy Diving Manual 1932). It should be noted that all oxygen stages of decompression were "in the dry" in a submerged decompression chamber with attendant. The reasons for these precautions are not widely known. In these trials even the experienced deep sea divers, who could work at 200 fsw, or somewhat deeper, on air, became confused, hallucinated and showed emotional instability at the greater depths (Damant, 1930). After long deliberation these alarming symptoms were attributed to the action of hyperbaric oxygen in the compressed air (up to 2.3 ATA). It was therefore concluded that breathing oxygen at over 33 fsw underwater was dangerous and that any oxygen stops should be in a chamber with an attendant (J.S. Haldane (père) & Priestley, 1935). In 1935 Behnke and his colleagues introduced the concept of nitrogen narcosis and subsequent developments, particularly oxyhelium diving, fully supported this thesis. However, with remarkable serendipity, the practice of all oxygen decompression stops being carried out "in the dry", even under water, continued in the Royal Navy.

After the large series of resting and working oxygen dives described in Chapter 2, it was concluded that even resting oxygen divers were at risk when deeper than 25 fsw (Donald, 1945). This work was not concerned with decompression but it was obviously relevant to the breathing of oxygen during in-water decompression stages. Behnke (1946) still felt, however, that if the diver was at complete rest then oxygen breathing during decompression at 60 to 40 fsw in water was safe. The US Navy continued to employ underwater oxygen decompression stages at 60 fsw (maximum 15 minutes) and 50 fsw (maximum 117 minutes).

The 60 fsw oxygen decompression stage was finally given up completely owing to a number of episodes of acute and severe oxygen poisoning while at this depth (Gillan, 1966). The maximal time at 50 fsw has now been reduced from 117 to 19 minutes. The 40 fsw stage now has a maximal time of

99 minutes. Both these maximal times are still far from safe but the US Navy precautions in case of acute oxygen poisoning are very thorough. The diver is supported by a submerged platform which guarantees a "resting state" and, in the event of oxygen poisoning, ensures immediate and certain retrieval. Divers must wear a rigid helmet or mask so that there is no risk of water inhalation if convulsions occur. Attendants, aware of the hazards of oxygen poisoning, are duly vigilant.

In the Royal Navy oxygen decompression stages from 60 fsw in the dry in submerged decompression chambers or inboard chambers continue to be used in routine diving. The only exception to this procedure is during decompression underwater of counter-mining oxyhelium divers. Neither surface decompression nor inboard nor outboard chamber decompression is possible as no diving vessel is allowed above the mine or mines in question, inflated rafts being used. These divers, for obvious reasons, always surface in as short a time as possible and oxygen stage in-water decompression is employed. The limit of dives are for 15 minutes at 230 fsw (70m) and thus the maximal oxygen stages are 3/50, 4/40, 5/30, 26/20. Even with these short stops at toxic depths, a number of instances of acute oxygen poisoning have occurred (Leitch, 1984), and these divers must now always work in pairs.

The lesson to be learnt from these experiences is that rest (quiescent state) does not give the remarkable protection from oxygen poisoning under water that has been claimed. The comparative tolerance at rest and on exercise at 30 to 50 fsw underwater, as already mentioned, has not been fully studied since the Royal Navy series in 1945.

Present Position

Divers are still exposed to known toxic tensions of oxygen while decompressing underwater. The occurrence of instances of oxygen poisoning, some unreported, has considerably reduced the degree of exposure as the years go by. In the United Kingdom sector of the North Sea, oxygen is not administered at depths greater than 40 fsw and the diver is always accompanied in the diving stage or wet bell by a standby diver. It is the policy of most diving companies that, while breathing oxygen, both divers are secured in the wet bell. Immediate air breathing facilities are available in all wet bells (large pocket of air or air breathing system).

Some well known Dutch diving companies now limit oxygen decompression in water to 9 m (30 fsw) and this is always in a wet bell with access to air breathing intermissions even at such shallow oxygen stops (Sterk, 1986). It is probable that in the next decade, the routine exposure of divers to known toxic tensions of oxygen while under water, will be challenged even more formally on ethical grounds. It is difficult to imagine any health and safety authority (if asked) giving permission for repeated routine exposure of divers to such tensions. However, as detailed above, the deeper oxygen decompression stages in water are being given up and elaborate precautions are being taken to protect divers decompressing on oxygen underwater, even at relatively shallow depths. Modern diving techniques and stricter safety standards may well ensure that decompression breathing oxygen underwater at depths below 20 fsw will soon become diving history.

REFERENCES

BARDIN, H. and LAMBERTSEN, C.J. (1970): A quantitative method for calculating pulmonary toxicity. *Inst. for Environmental Medicine Report, Philadelphia, University of Pennsylvania.*

BEHNKE, A.R., THOMSON, R.M. and MOTLEY, E.P. (1935): The psychological effects from breathing air at 4 atmospheres. *Amer. J. Physiol.*, 112, 554-558.

BOOTHBY, W.M. MAYO, C.W. and LOVELACE, W.R. (1939): Oxygen and oxygen and helium therapy: recent advances. *Med. Clin. N. Amer.*, 23 977-1005.

CALDWELL, P.R.B., LEE, W.L., SCHILDKRANT, H.S. and ARCHIBALD, E.R. (1966): Changes in lung volume, diffusing capacity and blood gases in men breathing oxygen. *J. Appl. Physiol.*, 21, 1477-1483.

CLARK, J.M. (1970): Derivation of pulmonary oxygen tolerance curves describing the rate of development of pulmonary oxygen toxicity in man. *Ph. D. Thesis, University of Pennsylvania.*

CLARK, J.M. and LAMBERTSEN, C.J. (1971a): Pulmonary oxygen toxicity, *a review. Pharm. Rev.*, 23 37-133.

CLARK, J.M. and LAMBERTSEN, C.J. (1971b): Rate of development of pulmonary oxygen toxicity in man during oxygen breathing at 2 ATA. *J. Appl. Physiol.*, 30, 739-752.

CLARK. J.M. (1982): Oxygen poisoning. *The physiology and medicine of diving.* 3rd Ed., Ed. Bennett and Elliott. Balliere Tindall, London. 200-239.

DAMANT, G.C.C. (1930): Physiological effects of work in compressed air. *Nature (Lond.)*, 122-6, 606-608.

DAMANT, G.C.C. (1951): An epic of treasure recovery. *Deep Diving, R.H. Davis, St. Catherine's Press, London.*, 358-372.

DONALD, K.W. (1944): The effects of breathing oxygen under pressure. *Alleviation of symptoms. cit. Admiralty Fleet Order 4565/44 para.6.*

DONALD, K.W. (1944): The variation of oxygen tolerance of human subjects in the wet and in the dry. *Admiralty Experimental Diving Unit Report XIII, UPS 40, R.N.P.R.C. 44/95.*

DONALD, K.W. (1945): Oxygen poisoning in man. *M.D. Thesis, Cambridge University.* Later issued as *Admiralty Experimental Diving Unit Report XVI/1946.*

DONALD, K.W. (1953): The definition and assessment of respiratory function. *Brit. Med. J.*, 1, 415 and 473 (see pp 450).

GILLAN, H.W. (1966): Oxygen convulsions in man (1966). *Proc. Third International Conference in Hyperbaric Medicine. Pubn. 1404, Nat. Ac. Sc. & Nat. Res. C., Washington.*

HALDANE, J.S. and PRIESTLEY, J.G. (1935): Respiration. *Clarendon Press, Oxford.*

HALL, D.A. (1967): The influence of the systematic fluctuation of PO_2 upon the nature and development of oxygen toxicity in guinea pigs. *Master's Thesis, University of Pennsylvania.*

HELVEY, W.M., ALBRIGHT, G.A., BENJAMIN, F.B., GALL, L.S., PETERS, J.M., and RIND, H. (1962): Effects of prolonged exposure to pure oxygen on human performance. *Republic Aviation Corporation Report 393-1, NASA Contr. NASr1-92.*

HENDRICKS, P.L., HALL, D.A., HUNTER, W.H. and HALEY, P.J. (1977): Extension of pulmonary oxygen tolerance in man at 2 ATA by intermittent oxygen exposure. *J. Appl. Physiol. Resp. Environ. Exercise Physiol.*, 42, 593-599.

LAMBERTSEN, C.J. (1955): Respiratory and circulatory actions of high oxygen pressure Proc. *Underwater Physiol. Symp. NAS/NRC, Publ. 377, Washington, D.C.*

LEITCH, D.R. (1984): Cerebral oxygen toxicity during immersion and surface supported diving. *Inst. of Naval Medicine (R.N), Technical Memo.* 12/84.

OHLSSON, W.T.L. (1947): A study in oxygen toxicity at atmospheric pressure. *Acta. Med. Sc. and Suppl.* 190.

STERK, W. (1986): Intermittent hyperoxia in operational diving. *Proc. Twelfth Annual Meeting. European Undersea Biomed.Soc.*, 52-64.

STERK, W. (1986): Personal communication.

STERK, W. and SCHRIER, L.M. (1985): Effects of intermittent exposure to hyperoxia in operational diving. *Proc. Twelfth Annual Meeting, European Undersea Biomed. Soc.*, 123-131.

WIDELL, P.J., BENNETT, P.B. KIVLIN, P. and GRAY, W. (1974): Pulmonary oxygen toxicity in man at 2 ATA with intermittent air breathing. *Aerospace Med.*, 45, 407-410.

WRIGHT, B.M. and McKERROW, C.B. (1959): Maximum forced expiratory flow rate as a measure of ventilatory capacity. *Brit. Med. J.*, 2, 1041-1047.

WRIGHT, W.B. (1972): Procedure for calculation of pulmonary oxygen toxicity. *US Navy Exp. Diving Unit Report. Proj. X-337, Report No.1.*

CHAPTER 5

THE DEVELOPMENT AND USE OF OXYGEN-NITROGEN MIXTURES IN DIVING

It is worth briefly reviewing the history of the use of artificial mixtures of gases in diving up to 1942. Paul Bert (1878) was the first to propose and demonstrate by experiments that the oxygen toxicity of various mixtures of nitrogen and oxygen was entirely a function of the partial pressure of oxygen being breathed. He also suggested the use of nitrogen and hydrogen to dilute the oxygen appropriately in deeper dives.

In 1912 Robert Davis and Leonard Hill devised a self-contained diving suit with rigid helmet for diving to depths of 100 fsw (Hill, 1912). The diver carried 50% oxygen-nitrogen mixture (ONM, the percentage given is always that of oxygen) in the two cylinders. The mixture was supplied at 5 1/min. (at 1 AT) and the flow of gas through an injector sucked the helmet air through the carbon dioxide absorbent canister and back to the helmet. J.S. Haldane (père) advised on the percentage of oxygen used. There were no relevant human experiments or observations on which to base his recommendations. The range of oxygen breathed at 100 fsw would have been of the order of 1.2 to 1.9 ATA, assuming constant mixture flow at increased pressure. As the tensions of nitrogen breathed were considerably less than when using air, decompression was greatly shortened. With moderate activity at 100 fsw the equivalent air depth would have been of the order of 40 fsw.

In 1923 J.S. Haldane (père) recommended a wider range of flow and oxygen fraction of the mixture allowing dives up to 150 fsw (Davis, 1951). As far as I know, these mixtures were neither tested nor used. Air diving was simpler and cheaper and even the first Davis-Hill suit was not generally adopted.

In 1919 Elihu Thompson proposed the use of helium-oxygen mixtures for diving on the assumption that the lower molecular weight and great

diffusibility of this gas would decrease the dangers of decompression sickness. United States Navy trials in 1924 were disappointing and the project was abandoned.

During the Admiralty Deep Air Diving Trials in 1930-33 up to 320 fsw, disabling retardation, mood changes and loss of judgement were encountered in large groups of men for the first time (Damant, 1930). It was not until 1935 that Behnke et al first proposed that these changes were due to nitrogen narcosis. They suggested that this could be remedied by using a rapidly diffusible, sparingly soluble gas with a low fat/water partition coefficient. Strangely, they did not specify helium.

End (1937) appears to be one of the first, if not the first, to report that there was a "marked absence of psychological change" breathing oxygen-helium mixture at considerable pressures. His colleague, Nohl, used such a mixture in a world record dive to 420 fw in Lake Michigan in 1937 (End, 1938). This truly remarkable dive had an almost "Alice in Wonderland" aura. Nohl's suit and apparatus was self-contained with mouthpiece and CO_2 absorbent canister. He only used "a respirable oxy-helium mixture" to distend his suit during descent. He controlled the flow of oxygen by hand "to satisfy his metabolic requirements" and "to maintain an oxygen partial pressure near to that of the atmosphere." End pointed out the great economy in the use of the expensive oxy-helium mixture and the ease with which the diver could administer increased oxygen to himself while in the water carrying out decompression stops. Indeed, in this particular dive, Nohl refilled his suit with oxygen when at his final 30 fsw stop.

At the beginning of this dive the diver fouled his telephone cable at 200-240 fw and was at or below 200 fw for 26 minutes. He surfaced in four minutes and re-entered the water two minutes later to dive to 420 fw. He thus practised an unintentional surface decompression type of dive (see Appendix 1) immediately before his main world record dive. While admiring the originality and ingenuity of these two men and the courage and skill of Mr Nohl, all the gods who protect divers were certainly present on that day.

Position in 1942

When the dangers of breathing pure oxygen underwater had been encountered and defined in 1942, a safer method of self-contained diving to

greater depths (30 to 120 fsw) was sought. An "intermission technique" was developed for possible use in covert operations of outstanding importance, where oxygen dives up to 60-70 fsw might be necessary (K.W. Donald, 1944). Short periods at full depth were alternated with brief intermissions at 10 fsw or less i.e. 10/60, 2/10, 10/60 etc., or 7/70, 5/10, 7/70 etc., (time in min/depth fsw). It was emphasised that should the diver develop symptoms during this procedure, he must come up at once to, or near, the surface and stay there as it had been found in the experimental oxygen exposures that a return to depth was highly dangerous. Although a very considerable number of experimental dives were carried out successfully the procedure was not considered to be entirely safe. More extensive trials would be necessary before any further operational application.

"Human Minesweeper" P-Parties. Oxygen-nitrogen mixture diver. Note raised counterlung in "ruff" position. Cylinder worn in "sword" position. Constant mass flow of mixture.

A far more satisfactory solution was found in a semi-closed counterlung breathing apparatus using oxygen-nitrogen mixtures (ONM) which was developed for the first time (Donald, 1943, (i)). As already noted, Paul

Bert first suggested that oxygen could be diluted with nitrogen to avoid the dangers of oxygen poisoning. A number of problems had to be solved. The increased flow of the gas mixture through the apparatus, which was essential, caused considerable discomfort. The counterlung was therefore worn round the shoulder (lower neck) and this, with improvements in the relief valve (larger diaphragm and stronger and more reliable spring), made the flow of mixture acceptable to the diver. A new reducing valve was produced to ensure a constant mass of gas supply independent of ambient pressure (expressed for convenience as 1/min at 1 bar). An equation was developed to enable the calculation of the optimal oxygen fraction and flow of the gas mixture with different ranges of depth and activity of the diver. J.B.S. Haldane (fils) gave valuable help. The essence of the equation was that, in a steady state, the partial pressure (pp) of oxygen in the counterlung breathed by the diver and in the gas vented from the counterlung is the same. Thus:

$$\text{pp oxygen breathed (bars)} = \frac{\text{Volume of oxygen vented}}{\text{Total volume of gas vented}} \times \text{Absolute ambient pressure (PAMB)}$$

$$\text{pp oxygen breathed (bars)} = \frac{\text{Mixture flow} \times \text{oxygen fraction} - \text{oxygen uptake}}{\text{Mixture flow} - \text{oxygen uptake}} \times \text{PAMB}$$

Calculations using this equation made it possible to avoid oxygen poisoning at maximal depth with minimal activity and dangerous hypoxia near the surface with maximal activity. An oxygen partial pressure of 2 ATA, then considered the safe limit, was never exceeded. In deeper dives the safety of immediate surfacing or the need for decompression stops had to be considered. One of the great advantages of the constant mass mixture supply is that, if the diver's oxygen uptake rises, owing to increased exertion, the partial pressure of oxygen being breathed is reduced. As exercise increases the risk of oxygen toxicity, the associated fall of inspired oxygen tension was a most important safety factor at greater depths.

A large series of experimental oxygen-nitrogen mixture dives was carried out to different depths and with different degrees of activity and

oxygen uptake, first in dry chambers and then underwater. Gases were sampled from the counterlung during various stages of these dives to check against calculated figures and to be sure of the partial pressure of oxygen and nitrogen being breathed in each experimental dive. Both non-toxic and deliberately toxic equivalent oxygen depths were tested in underwater exposures. The same divers carried out pure oxygen breathing under water at the same partial pressure of oxygen before and after such dives but, with the known variation of oxygen tolerance in the same individual, these could hardly be called controls. However the strong impression gained was that *"the depth limits of pure oxygen diving could be safely applied to the equivalent oxygen depth in oxygen-nitrogen mixture diving"* (Donald, 1943, (i)).

In these investigations the rate of achievement of a steady state (as regards gas tensions in the counterlung) during vigorous exercise (simultaneous trotting on the spot and weight lifting) was studied at 20, 40, 60 and 80 fsw in compressed air. As was anticipated, the "buffering" action (as regards PO_2 levels) of the gas in the counterlung, apparatus and diver's lungs was much more marked and prolonged at the greater pressures. This emphasized that with minimal "buffering" the risk of hypoxia during hard work at shallow depths was considerable. It also meant that, as very hard work is rarely sustained underwater by booted divers, the oxygen levels were higher and nitrogen levels lower than those calculated for the steady state. This was therefore favourable with regards to decompression sickness at the greater depths. (Donald, 1943, (i)).

From the above equation and considerations it was abundantly clear to us that an accurate knowledge of the divers' oxygen uptake was critically important. At first the somewhat crude method of balancing oxygen supply and uptake while in a steady state of rest or exercise was used. A far more accurate and widely applicable method, using a demand valve in the counterlung with precise monitoring of the pressure fall in a small cylinder at surface, was developed. (Donald and Davidson, 1944). Measurements were made of the oxygen uptake of booted and fin-swimming divers in a great variety of activities and environments (depth, tide, mud, etc).

We were able to measure the range of oxygen uptake of divers carrying out specific tasks such as Port clearance (P parties) booted divers during actual searching on a grid in harbours and basins at depths up to 80 fsw.

This allowed us to use a fairly low flow (3 1/min) of 65% oxygen-nitrogen mixture (the percentage given is always that of oxygen) for these divers as there was no marked near-surface activity and known degrees of oxygen uptake while searching on the bottom. Endurance and economy of mixtures were very important considerations under severe operational conditions. In the unusual instance of a P party oxygen-nitrogen counterlung diver working in significant tides or very heavy mud, flow could be increased.

Experimental booted diver wearing "oxygen uptake" breathing apparatus. Small oxygen cylinder with accurate pressure gauge held by attendant at surface. Pressure tubing carrying intermediate pressure (48 psi) to demand valve in divers counterlung. Soft helmet air diver (Donald's wonder) also in attendance.

No oxygen poisoning, decompression sickness or hypoxic episodes were reported in thousands of operational dives in Europe.

Dutch, Norwegian and Belgian divers were trained in P party mixture diving technique during and after World War II.

For counter-mine diving in deeper tidal water an improved form of the original Davis-Hill self-contained rigid helmeted suit was being adopted (1943). A 6 1/min flow of 60% oxygen-nitrogen mixture was employed but, owing to the recently appreciated dangers of oxygen poisoning, dives deeper than 70 fsw were not allowed.

Mine Recovery Suit. Mixture breathing. Self-contained. Note robust helmet, suit and boots for heavy work on bottom in tidal waters.

Mine Recovery Suit. Mixture breathing. Rear view showing three gas cylinders (one emergency), reducing valve leading to venturi-injector and carbon dioside absorbent canister. Venturi sucks gas from helmet (no mouthpiece), through canister and back to helmet.

It was imperative to be able to go deeper to at least 120 fsw and the Admiralty Experimental Diving Unit were asked for advice, not only with regard to oxygen poisoning and decompression sickness, but also to the possibilities of accelerated or immediate surfacing. We were able to carry out a large series of investigations. The "mixture equation" had never been applied to this apparatus nor had any studies been carried out in open water or pressure chambers. Many anti-mining personnel acted as subjects and attendants.

After due calculations and measurements of oxygen uptakes during different tasks a 45% oxygen-nitrogen mixture with a flow of 8 1/min was proposed for dives up to 120 fsw. A series of working dives was carried out with decompression stages, when necessary, according to the equivalent air depth. This was easily calculated from the partial pressure of nitrogen in the mixture being breathed. Equivalent oxygen depth with minimal activity never exceeded 2 ATA.

It was appreciated that the Royal Navy air decompression tables were rather generous in this depth range and we shortened the decompression stages even further without any untoward events. Emergency "immediate surfacing" was also carried out after an hour on 45% oxygen-nitrogen mixture at 85 fsw and one hour at 100 fsw (moderate work). There was considerable itching and some minor transient bends not requiring recompression. In the report on this work (Donald, 1943, (ii)), the author stated "These rather heroic performances are emphatically not recommended as a routine. The fact that they are possible in an emergency, however, is a great consolation to personnel diving on mines, even though there is a definite risk of bends, apparently slight, unless hard work is done." Again, no oxygen poisoning or decompression sickness was reported during subsequent counter-mining operations throughout Europe in 1944 and 1945.

Reverting to counterlung mixture diving, for more general purposes, a flow of 4 1/min. of 60% oxygen-nitrogen mixture was used to dive up to 80 fsw. These divers could carry out a wider range of activities but were still booted and never wore fins.

The possibility of "frogmen" (oxygen breathing underwater fin swimmers) using oxygen-nitrogen mixture was considered at an early stage. When their oxygen uptake was measured during underwater swimming it was found to be surprisingly high (Donald and Davidson, 1944). Higher

flows of mixture would have been required and this would have greatly increased venting and reduced endurance. As all "frogmen" were in covert operations and did not swim below 25 fsw, it was considered prudent to continue the use of oxygen.

Rigid helmeted self-contained mixture diver being lowered into the "wet pot".

Oxygen Bends

The experiments described (Donald, 1943 (i & ii)) made it clear that, just as nitrogen had no effect on oxygen poisoning, the oxygen (up to 2 ATA) had no important effect on the absorption and elimination of nitrogen, nor did it contribute significantly to bubble formation. In other words, with the mixtures used, the partial pressure of nitrogen breathed was a safe measure of the decompression required. However, in view of the possibility of even deeper oxygen-nitrogen mixture dives and the possible use of hyperoxic mixtures in submarine escape, the author felt that the possibility of raised oxygen tensions contributing to decompression sickness should be tested to the maximal degree (Donald, 1945).

The following procedure was adopted. First it was confirmed that goats had no decompression sickness after immediate decompression following one hour breathing air at 50 fsw (1.5 ATS) or after breathing oxygen for one hour (82%) at 110 fsw (4.3 ATS). They were next compressed to 50 fsw using air and then, immediately, to a further 100 fsw using oxygen. They were now at 150 fsw breathing 62% oxygen. The equivalent air depth was 50 fsw and the equivalent oxygen depth 82 fsw. After one hour at this pressure they were decompressed (1.25 ft./sec) to one atmosphere of air. This caused chokes (pulmonary oedema with vascular bubble embolism), paralyses (transverse myelitis) and multiple bends. Then, after 10 to 25 minutes at one atmosphere, quite dramatically and uniquely, all these mortal signs of decompression sickness resolved completely and the animal returned to total normality. One goat (out of 7) did not appear to be recovering satisfactorily and was cured by therapeutic recompression. Thus an additional 3.0 atmospheres of oxygen had caused extremely severe decompression sickness. The spontaneous resolution of the condition without recompression confirmed that the bubbles were mainly oxygen and that they were duly removed by body metabolism. Unfortunately further experiments to determine the minimal added tension of oxygen to 50 fsw air to precipitate transient decompression sickness could not be pursued at this time.

Oxygen-nitrogen mixture divers operate at depths up to 140 fsw (42m) and occasionally to 180 fsw (55m). The maximal oxygen partial pressure in Royal Navy mixture dives has always been 2 ATA. As far as I know, there have been no reported instances of decompression sickness when the equivalent air depth and appropriate decompression has been assessed from the nitrogen partial pressure.

These findings indicated that the maximal partial pressure of oxygen that can be safely added to a tolerable nitrogen overload during immediate decompression lies between 2.0 and 3.5 bars. It would still be of great interest to define this level more precisely. It was also evident that the rigidly imposed maximal level of 2 ATA partial pressure of oxygen in oxygen-nitrogen mixtures, to avoid acute oxygen poisoning, had also acted, quite unbeknown to us, as a most effective safeguard against another potentially dangerous condition of acute "mixed hyperoxic decompression sickness."

Mixture Diving 1945 to Present

After World War II the use of semi-closed counterlung mixture diving was continued and considerably expanded in the Royal Navy. Fin swimming divers undertook many of the tasks previously performed by the classical rigid helmeted and booted divers including a great deal of countermining activities. Many divers now used air-on-demand open circuit apparatus (aqualung type). Others used counterlung oxygen-nitrogen mixture diving which, with constant mass flow, made far more economical use of the gas carried and allowed longer and deeper exposures without decompression sickness.

The oxygen-nitrogen mixtures used in self-contained counterlung diving have not changed greatly in the last 50 years. The standard NATO mixtures and mass flow rates for various depth ranges were derived from the original Royal Navy work described above and are as follows:

Mixture B: 60/40 oxygen-nitrogen, flow STPD 4 1/min, depth limit 25m (80 fsw).

Mixture C: 40/60 oxygen-nitrogen, flow 8 1/min, depth limit 42m (140 fsw).

Mixture D: 32.5/67.5 oxygen-nitrogen, flow 12.8 1/min, booted divers only, depth limit 55 m (180 fsw).

For some years the Royal Navy has increased the recommended flow by 50% when swimming under water (6 & 12 1/min. of mixtures B and C respectively). Recently this maximal flow has been adopted for all purposes, another indication of the increasing dominance of the fin-swimming diver with his higher oxygen uptake.

It will be noted that the maximal partial pressure of oxygen, with minimal oxygen uptake at maximal depth, with these NATO mixtures and flows is still 2 bars.

The extreme secrecy concerning the use of oxygen-nitrogen mixtures in World War II, particularly in countermining activities, was so effective that for a number of years many authorities were not aware of its existence. As late as 1969 an eminent physiologist reporting on animal experiments with oxygen-nitrogen mixtures in Acta Physiologica Scandinavica concluded that his "results indicated that it should be

worth while to start experiments on human beings with oxygen-nitrogen mixture as diving gas to develop a non-decompression diving technique for a depth down to 23 m."

Lanphier (1954), when developing oxygen-nitrogen counterlung mixture diving in the US Navy for the first time, wrestled independently with exactly the same problems as those encountered and solved by the Royal Navy in 1943 i.e. the calculation of gas tensions in the diver's circuit (mixed gas equation), the oxygen uptake of divers and swimmers, the provision of constant mass flow of gas mixture and the effectiveness of applying the "principle of partial pressure" in assessing the risk of oxygen poisoning and decompression sickness.

In testing the safety or otherwise of the equivalent oxygen depth while breathing oxygen-nitrogen mixture, Lanphier (1955, (i)) began with a working dive on 47% ONM at 100 fsw, using an open circuit system. The equivalent oxygen depth was 29 fsw. The diver convulsed after 20 minutes. Lanphier referred back to his "pure oxygen" tolerance test curve (Lanphier & Dwyer, 1954) at this depth, where eleven subjects had completed 58 minutes without symptoms (see Fig. 19, Chapter 3). However his working limit curve time was 46 minutes. He observed (Lanphier, 1955, (i)) "Not even the well known swings of individual susceptibility could explain this convulsion adequately", he continued, "subsequent exposures produced enough other symptoms to indicate we were contending with reality." No individual details were given. He concluded "a given partial pressure of oxygen was simply proving more toxic in the presence of increased nitrogen pressure."

The oxygen-nitrogen mixture dives referred to above were not published but there is a description of them in USNEDU Report 7-55. 15 subjects carried out working ONM dives on an unspecified number of occasions to depths between 90 and 140 fsw, breathing 40 to 49% oxygen. The range of equivalent oxygen depth (DO_2) was 16 to 37 fsw. An open circuit was used with the diver swimming against a trapeze (8lb. spring). VO_2 was of the order of 1.5 l/min.

Unfortunately particulars are only given of the dives during which the subjects developed symptoms. It is not stated whether the dives continued after symptoms had occurred nor is the total duration of any dive recorded. The number and types of ONM dives without symptoms by the individual divers are not reported.

The subject CLL, who convulsed in the first dive of the series (DO_2 29.5 fsw), suffered nausea in another ONM dive (DO_2 16 fsw) after 45 minutes. Of the remaining 14 subjects three complained of nausea at 23, 35 and 36 minutes, all at a DO_2 of 24 fsw. One of these subjects in another dive, at a DO_2 of 36 fsw, complained of severe generalised tremors (shivering) after 4 minutes. Two other subjects complained of vertigo, in one case "transient and early" at a DO_2 of 37 fsw and in the other after 16 minutes at a DO_2 of 24 fsw.

In five instances divers also complained of non-specific symptoms such as headache and fatigue, with or without other symptoms. Finally, six of the 15 divers had no symptoms during an unspecified number of mixture dives of unspecified duration with DO_2 ranging from 16 to 37 fsw.

The nausea and vertigo occurring during these mixture dives with DO_2 from 24 to 37 fsw were presumably due to oxygen poisoning. Lanphier compared these episodes with those in his preliminary working oxygen diving studies (Lanphier & Dwyer, 1954) where in 5 exposures to 35 fsw (max. 43 mins), one subject convulsed in 42 minutes and another suffered nausea after 28 minutes. In 11 exposures to 30 fsw (max. 57 mins), and in 5 exposures to 25 fsw (max. 81 mins), there were no symptoms. Although the number of observations was not adequate, particularly at 25 and 35 fsw, it was reasonable to suspect from his own limited findings that oxygen might be more toxic when breathed in oxygen-nitrogen mixtures at pressure.

Lanphier took no account of the Royal Navy oxygen working dives (1944-6) from 40 to 25 fsw, where considerable oxygen poisoning was encountered, including a convulsion after 10 minutes at 30 fsw. Even in resting dives at these depths there was considerable toxicity including a convulsion after 30 minutes at 30 fsw (see Tables 18, 21, 22 and 23 in Appendix 2). The reader is reminded that some of these R.N. subjects were selected for this series because of their known low average oxygen tolerance and that all dives were in fact, 2 to 3 fsw deeper than the gauge reading.

Nevertheless these R.N. findings should have given rise to some pause but Lanphier concluded from his rather slender evidence that oxygen toxicity was increased in oxygen-nitrogen mixtures. He did express some reservations in so far as he recommended "direct studies of

oxygen tolerance during exposure to nitrogen-oxygen mixtures at depths approaching the limits of tolerance more closely than before" (Lanphier, 1955). Such studies, as far as the author knows, were never carried out. Thus his total evidence that, contrary to the Royal Naval findings (Donald, 1943 (i) & (ii)) and experience, oxygen was more toxic when breathed in oxygen-nitrogen mixtures is of little formal significance.

It will be recalled that Dickens (1945), in tissue respiration studies, found that increased nitrogen tensions exerted neither a protective nor a deleterious effect on oxygen poisoning as manifested by the respiration of rat brain slices.

Apart from the effects of increased tensions of nitrogen on oxygen poisoning, Lanphier considered other possible causes of accelerated oxygen toxicity. He made the important point that the density of the gas mixture at depth (say 99 fsw) is twice that of the density of oxygen at, say, 30 fsw. Thus the increased respiratory resistance (40% approx. greater), both internal and external, might cause reduced ventilatory volumes and a resultant rise of alveolar and body carbon dioxide levels, particularly when exercising. He also thought it possible that certain individuals had a reduced sensitivity to carbon dioxide under these conditions and continued to tolerate the increased carbon dioxide levels without an appropriate ventilatory response. Increased body carbon dioxide tensions would certainly make the diver far more prone to oxygen poisoning (Hill, 1933).

Lanphier investigated these possibilities and his findings and conclusions are reviewed at some length in Chapter 6. Suffice to say here that the author feels that much more evidence is needed to prove that an important degree of carbon dioxide retention occurs in operational counterlung mixture dives. Further the total lack of oxygen poisoning in such divers in the Royal Navy and later in NATO over half a century, is powerful but indirect evidence to the contrary.

However the US Navy appeared to have been convinced and even alarmed by these considerations and the following severe restrictions were placed on the partial pressure of oxygen and time exposure when oxygen-nitrogen mixture diving was first introduced (1959).

US NAVY OXYGEN PARTIAL PRESSURE LIMITS IN OXYGEN-NITROGEN MIXTURES

NORMAL EXPOSURE		EXCEPTIONAL EXPOSURE	
PO_2 (Bars)	Time (min)	PO_2 (Bars)	Time (min)
1.6	30	2.0	30
1.5	40	1.9	40
1.4	50	1.8	60
1.3	60	1.7	80
1.2	80	1.6	100
1.1	120	1.5	120
1.0	240	1.4	180
		1.3	240

Thus the limit of partial pressure of oxygen in oxygen-nitrogen mixture diving was now 1.6 ATA, equivalent to an oxygen depth of 20 fsw. Time limits were also given from 30 minutes at 1.6 ATA to 240 minutes at 1.0 ATA. These time limits appear to have been quite arbitrary and unrelated to acute or pulmonary oxygen poisoning. The "exceptional exposures" (emergency or covert) time limits were not dissimilar to the standard US Navy working limits for pure oxygen at that time. These restrictions cause a considerable limitation in the scope of mixture diving, even more so now that the maximal partial pressure of oxygen allowed has been further reduced in the US Navy to 1.4 ATA (13 fsw on pure oxygen).

Other Types of Counterlung Mixture Breathing Apparatus

The Constant Ratio Breathing Apparatus (DC55) is a well known counterlung apparatus used for mixture diving. It was first employed by the French Navy and adopted for military and commercial purposes in many other countries. The mixture usually breathed is 60% oxygen-nitrogen mixture. The counterlung is in the form of a concertina bellows. Inside this is a smaller slave bellows which follows its movements faithfully. The ratio of volume of slave to main bellows is constant (1/11). When the diver exhales he fills both bellows, when he inhales the expired gas in the slave bellows is vented to the sea. The larger bellows is fed by a demand valve, through intermediate pressure, from the mixture 'bottles' and thus the volume of gas in the counterlung is maintained.

This apparatus, despite its remarkable ingenuity, has some disadvantages. Firstly, the amount (mass) of mixture vented and therefore demanded is a direct function of the diver's ventilation (1/11) and also of the diver's depth. The endurance of the set when swimming at any significant depth is therefore greatly reduced. The second disadvantage is even more important and not always appreciated. The mixed gas equation, developed by the Royal Navy in 1943, can still be used to calculate the partial pressure of oxygen being breathed by the diver in this type of circuit (Williams, 1975). The "flow" of gas mixture in this case is the volume demanded on inspiration. As loss and gain of mixture are balanced, this is equal to the volume vented (1/11 of expired gas) plus the volume of oxygen used by the diver. Using the 60% oxygen-nitrogen mixture, it can be calculated that:

$$pp\,O_2 = 0.6 \times PAMB - 0.4/kr$$

Where PAMB = ambient pressure, k = Ventilatory equivalent (Ventilation per unit O_2 used) and r = bellows ratio = 1/11.
The equation is not very sensitive to the usual range of the ventilatory equivalent. Assuming k = 22, we have therefore:

$$pp\,O_2 \text{ breathed (approx.)} = 0.6 \times PAMB - 0.2.$$

The partial pressure of oxygen breathed is thus almost directly proportional to the depth of the diver, no matter what degree of exercise is involved. For example at 5m the partial pressure of oxygen breathed is 0.7 bar and at 25m it is 1.9 bar. Fast swimming at 25m at an equivalent oxygen depth of 30 fsw (1.9 bar) could be hazardous as severe oxygen poisoning, including convulsions, has been encountered in working oxygen dives at 30 fsw (1.9 bar) and 25 fsw (1.75 bar) (Donald, 1945; Butler and Thalmann, 1986). Although this apparatus has been widely used for a number of years the almost direct relationship of depth and partial pressure of oxygen breathed is not generally appreciated.

A modification of DC55 used by the Swedish Navy regains some of the advantages of constant mass supply of mixture. A predetermined "dose" (mass) of the gas mixture is supplied at a frequency controlled by the ventilation. This "dose" can be calculated from the "gas mixture equation".

The supply of mixture is thus independent of depth but not of ventilation. Thus, again, if the diver exercises at or near maximal depth, his ventilation, mixture supply and inspired partial pressure of oxygen will rise in contrast to the fall of oxygen concentration that would occur in a constant mass flow apparatus.

The increasing complexity of these and other new forms of apparatus used in mixture breathing is in sharp contrast to the extreme simplicity of the original British counterlung mixture breathing apparatus. This simplicity ensures reliability, easy maintenance and durability. The protective effect of exercise allows safer access to greater depths without oxygen poisoning.

A closed circuit mixture breathing apparatus with oxygen sensors and automatic feed of oxygen and diluent gas has long been desired. With accelerating technological advances, a constant partial pressure of oxygen can be maintained with changing activities and depths. The oxygen tension can be programmed for safe but increased levels of oxygen during decompression when it is necessary. Relatively "simple" forms of such apparatus would allow the breathing of gas mixtures with little or no venting and pure oxygen diving, with its ever present dangers, would no longer be needed for covert operations. Various types of such apparatus have been successfully developed in several countries. Absolute reliability and trust is essential and it will take time to establish this. A further problem is that the sensors and associated circuitry could be vulnerable to a variety of high energy defensive sweeps.

Open Circuit Mixture Breathing. A most important development in recent years has been the increasing use of oxygen-nitrogen mixtures by SCUBA divers in place of air when using open circuit demand apparatus. For example, by this means a diver breathing 50% ONM can dive down to 80 fsw with relative impunity (maximal DO_2 25 fsw; maximal equivalent air depth 40 fsw). This is obviously advantageous in many diving tasks but without, or even with, supervision the practise is full of potential danger to an independent diver. For instance, if the air diver carries out an emergency dive or an accidental or fool-hardy excursion to greater depths, there is little risk of decompression sickness providing the diver returns to surface more cautiously. However, if he is using a hyperoxic mixture with an open circuit, such an excursion might cause acute oxygen poisoning and drowning, particularly as there has been a preceding hyperoxic exposure of

25 or more fsw. Again if a counterlung constant mass mixture diver uses his usual mixture with an open circuit he may not appreciate or remember the greater risk of oxygen poisoning when swimming or working hard near the maximal depth.

Open Circuit, Surface Demand, Mixture Breathing: The gas can be supplied from the surface or bell to the diver's demand valve by a hose holding gas at intermediate pressure, (as in Donald & Davidson, 1944). This technique not only increases endurance but ensures more effective supervision.

The Outstanding Problem:

Finally let us return to the important problem with regard to the safe limit of oxygen partial pressure in the use of oxygen-nitrogen mixtures. It is a remarkable fact that the two navies, both with considerable reputations in hyperbaric work, have had totally different criteria of safety in oxygen-nitrogen mixture diving over a period of thirty years. It is worth briefly recapitulating the reasons for this difference.

The Royal Navy could find no evidence, in a series of experimental oxygen-nitrogen mixture dives, that increased tensions of nitrogen altered the toxicity of oxygen (Donald, 1943 (i & ii). An oxygen partial pressure limit of 2 ATA, the then accepted limit for pure oxygen diving (1943), was therefore used for all oxygen-nitrogen mixture diving throughout the Second World War. This limit has continued to be used from that time and no instance of oxygen poisoning has ever been reported in oxygen-nitrogen mixture diving with constant mass flow in over 40 years. Even when 25 fsw was recommended (Donald, 1945) and adopted as the depth limit for pure oxygen diving, the partial pressure oxygen limit in oxygen-nitrogen mixture diving remained unaltered at 2 ATA. This decision was, no doubt, due to the known "protective" effect of exercise (reducing oxygen partial pressure) in the British oxygen-nitrogen counterlung apparatus and, even more importantly, to the total absence of oxygen poisoning in such mixture diving.

Nevertheless in many new types of counterlung oxygen-nitrogen breathing apparatus with *variable* mass of mixture supply, the protective effect of exercise is lacking and the oxygen tension breathed is a function of ventilatory volumes and often, of the depth as well (see above). For this

reason it would be prudent when using such apparatus, to lower the oxygen pressure limit considerably.

The United States Navy. Lanphier (1955 (i)), while developing the use of oxygen-nitrogen mixture diving in the US Navy, encountered an unexpected and dramatic convulsion in the first experimental dive of the series. This and some other mild episodes in these experiments (see above) led to his claim that oxygen was more toxic in the presence of hyperbaric nitrogen. This is entirely contrary to the Royal Navy's research and experience. Lanphier's "nitrogen" hypothesis has never been put directly to the test in thirty years. All that would have been required would be a series of (say) 60% oxygen-nitrogen mixture exercising dives with the subjects breathing oxygen partial pressures of (say) 1.6, 1.8, 2.0, 2.2 and 2.5 ATA. The same groups would then dive to equivalent depths on pure oxygen. Dives would need to be to end-point under carefully controlled wet chamber conditions. To allow for intra and inter-individual variation of oxygen tolerance, the groups used must be of adequate size (for example see Figure 19, Chapter 3 on the effect of exercise on oxygen tolerance). It is realised that experimental group diving on oxygen to end-point is now rarely, if ever, practised. The need to conduct such a series is not only because of the severe restriction of oxygen-nitrogen diving by certain schools but, even more important, *to eliminate a quite unnecessary ambiguity which should have been resolved many years ago by those responsible for the safety of divers.*

Lanphier has advanced a second hypothesis concerning possible carbon dioxide retention in oxygen-nitrogen mixture dives, to further explain the "unexpected dangers of oxygen poisoning" in such divers. The evidence to support this hypothesis is fully examined in the following chapters.

This chapter has been largely concerned with mixture diving by the armed forces. Their strict discipline and generous operational staffing are particularly favourable to the safety of mixture diving as is witnessed by their remarkable record over half a century. The adoption of counterlung mixture diving in commercial work caused some initial concern with regard to the standards of apparatus maintenance, supervision and procedure. However, on the whole, a high safety standard has been achieved by the industry. Nevertheless commercial divers and their teams are, on occasions, hard pressed over considerable periods and lapses of supervision

are possible under such conditions. Some of the factors increasing the danger of oxygen poisoning are the use of *variable* mass flow of mixture in new types of counterlung apparatus, the casual use of an open circuit in mixture diving, any undue and undetected variation of mixture composition or flow and finally, depth errors.

A number of commercial organisations have reduced the maximal permitted partial pressure of oxygen by limiting depths or changing mixtures to considerably increase the margins of safety. This was a wise and practical step. However the proposition that oxygen toxicity is significantly increased when breathing oxygen-nitrogen mixtures infers that there is an unquantified component of these excellent safety margins. It is critically important that this hypothetical danger, if it exists, should be properly measured in the same conclusive manner as were the effects of immersion and exercise on oxygen poisoning.

Finally there is the topical controversy concerning the use of oxygen-nitrogen gas mixtures by recreational divers. In view of the very high degree of training and constant detailed supervision required, one's first reaction is to have considerable doubts about such a proposal. Admittedly, there are many highly sophisticated groups (archeologists, biologists, photographers and so on) who could well satisfy the following requirements:

(1) The whole *team* to be scrupulously trained and tested in the theory and practise of mixture diving, both individually and working together. Duties and responsibilities should be largely interchangeable.

(2) During each and every diving session there should be (say): two divers (buddies), one stand-by diver, one diving supervisor on the surface who is in charge, supported by two other members who are also fully responsible for care, maintenance and checking of all apparatus including gas cylinders and their contents. Special attention must always be given to the constant mass flow reducing valve (sealed with jet orifice) which must be checked by a reliable and frequently calibrated flow-meter. It is also a long-standing tradition that all mixture divers carefully re-check their own diving apparatus (apart from instrumental checks) before each dive.

(3) Full knowledge must be obtained of the significance and possible risks of new variations of mixture breathing apparatus (particularly variable mass flow) coming on to the market.

(4) There must be an absolute depth limit with each particular gas mixture, remembering that breaking this limit is not just a matter of a possible bend but of convulsions and drowning. If possible, divers should carry a depth alarm signal (acoustic or vibrations).

(5) Changes in personnel in the group must be reported and approved.

Without in any way belittling the remarkable know-how and skill of many recreational divers, the maintenance of such a group at all times and for all dives could present difficulties. A reduction in the number of such a team could lead to excessive over-lapping of duties and possible brief but dangerous lapses of diver or apparatus supervision. If such groups were approved and registered by an appropriate authority it must be on the clear understanding that it is a complete working group that is being considered and never an individual diver or pair of divers.

The above conditions for mixture diving may seem to be quite unreasonably inhibiting and stuffy (even to the author as he writes). However, few would deny that no reputable organisation or service would depart, to any important degree, from such conditions in mixture diving. Again, without these or fairly similar conditions, adequate insurance and reasonable legal protection would not be possible.

REFERENCES

BEHNKE, A.R., JOHNSON, F.S., POPPEN, J.R. and MOTLEY, E.P. (1935). The effect of oxygen on man at pressures from 1 to 4 atmospheres. *Amer. J. Physiol.*, 110, 565-572.

BERT, P. (1878): *La Pression Barometrique. G. Masson, Paris.*

DAMANT, G.C.C. (1930): Physiological effects of work in compressed air. *Nature, London.*, 126 (2) 606-608.

DONALD, K.W. (1943, (i)): Low supply of mixtures in counterlung type of breathing apparatus. *Admiralty Experimental Diving Unit (R.N.) Report VII/43.*

DONALD, K.W. (1943 (ii)): The use of mine recovery suit at greater depths. *Admiraly Expermental Driving Unit (R.N.) Report VIII/43*

DONALD, K.W. (1944) cit. Admiralty Fleet Order 4565/1944

DONALD, K.W. (1945): Oxygen Bends. M.D.Thesis Cambridge University, 154-163. Later published DONALD, K.W. (1955) Oxygen Bends. *J. Appl. Physiol.*, 7, 639-644.

DONALD, K.W. and DAVIDSON, W.M. (1944): Oxygen uptake of divers. *Admiralty Experimental Diving Unit (R.N.) Report XV/44.* Later published: DONALD & DAVIDSON (1954). Oxygen uptake of booted and fin swimming divers. *J. Appl. Physiol.*, 7, 31-37.

END, E. (1937): Rapid decompression following inhalation of helium and oxygen mixtures under pressure. *Amer. J. Physiol.*, 120, 712-718.

END, E. (1938): The use of new equipment and helium gas in a world record dive. *J. Ind. Hyg. and Tox.*, 20, 511-520.

HILL, L. (1912): Caisson Sickness. *Edward Arnold, London & Longmans & Green, New York. Pages* 144-145.

HILL, L. (1933): Carbon dioxide and oxygen poisoning. *Q. J. Exp. Physiol.*, 23, 49-56.

LANPHIER, E.H. (1954): Gas mixtures in self-contained diving. U.S.N. *Experimental Diving Unit Special Report Series on diving with SCUBA., Chapter 6.*

LANPHIER, E.H. (1955, (i)): The use of nitrogen-oxygen mixtures in diving. *Proc. First Symposium Underwater Physiol., Pubn. 377 Nat. Ac. Sc., & Nat. Res. C., Washington D.C.*, 74-78.

LANPHIER, E.H. (1955, (ii)): Nitrogen-oxygen mixture physiology. *Phases 1 and 2. U.S. Navy Experimental Diving Unit, Washington, Report 7-55.*

THOMPSON, E. (1927): Copy of 1919 letter to Bureau of Mines suggesting use of helium in diving. *Science.*, 65, 36-38.

WILLIAMS, S. (1975): Engineering principles of underwater breathing apparatus. *Physiology & Medicine of Diving. Ed. Bennett, Elliott, Ballière Tindall, London.*

CHAPTER 6

CARBON DIOXIDE AND HYPERBARIC OXYGEN

Leonard Hill (1933) showed that relatively low tensions of carbon dioxide (5% 1 ATS) markedly increased the toxicity of hyperbaric oxygen at 4 ATA in monkeys, goats, guinea pigs and rats. It was known at that time (Forbes, 1928; Wolff and Lennox, 1930) that the inhalation of moderate concentrations of carbon dioxide caused dilatation of the pial arteries and, in 1935, Gibbs, Gibbs and Lennox showed that human cerebral blood flow was increased by raised alveolar carbon dioxide tensions. It was generally assumed therefore that, in these hyperoxic conditions, the carbon dioxide caused cerebral vasodilatation and increased blood flow causing a critical rise of brain tissue oxygen tensions.

Thus when, in 1942, the Royal Naval human torpedo oxygen divers (booted) suffered acute oxygen poisoning in times and at depths previously considered to be safe (40 to 70 fsw), the first suggestion was that these attacks were accelerated by increased tensions of carbon dioxide due to inadequate absorption or even "inefficient breathing" (Leonard, 1942). However this was soon shown not to be the case as analysis of the inspired gas of these divers under different operational conditions demonstrated extremely efficient carbon dioxide absorption and very low levels of inspired carbon dioxide (Donald, 1945). Nevertheless a new and unexpected hazard presented a few months later.

Shallow Water Black-Out

A number of unexplained cases of loss or marked disturbance of consciousness were encountered in underwater oxygen swimmers when diving at shallow depths (0 to 20 fsw). The series of events varied, but not greatly. These underwater swimmers (frogmen) described how they "were going away", "became muzzy" or "hazy", "everything went in waves"

"were confused" and so on. Some individuals reported that the sensation was "quite pleasant". A few divers also reported some respiratory distress but this was more the exception than the rule. No convulsions or any other signs or symptoms of oxygen poisoning were reported or seen. No apprehension was experienced. Some divers lost consciousness underwater while swimming and others surfaced and were hauled inboard in a highly dissociated condition. Recovery was rapid and normality regained in a few minutes. A number complained of nausea and severe headache after the event. The syndrome was somewhat similar to the early stages of induced general anaesthesia. The only important difference was a marked tremor during the first few minutes of recovery.

The possible causes of this dangerous phenomenon were investigated at the Admiralty Experimental Diving Unit and the National Institute for Medical Research (Barlow and MacIntosh). Data, reports and valuable opinions were obtained from the medical and executive officers of the various operational parties.

The following possible causes of these black-outs were considered:

Circulatory: Fainting attacks in fit young men are usually due to prolonged maintenance or sudden assumption of the erect posture. Standing still after marked leg exercise can also cause fainting. In the case of divers, if they are standing, the water pressure gradient will reduce any tendency to distribute excessive blood to the lower body and legs and, in any case, will favour cerebral circulation. If they are swimming and nearly horizontal such syncopal attacks would be even more unlikely.

The lung circulation and filling of the heart can be impeded by high sustained intra-pulmonary pressure, with a resultant fall of cardiac output and cerebral circulation. It is possible to produce loss of consciousness by expiring forcibly against a resistance particularly after hyperventilation has reduced body carbon dioxide and caused cerebral vasoconstriction (the well known "mess trick"). A sustained increase of pressure in the lungs and apparatus with rapidly reducing depth and gas expansion is not likely with an open glottis and a properly functioning exhaust valve. Swimmers and divers in the critical stages of covert operations shut their exhaust valve to avoid venting but they were scrupulously trained to reduce depth cautiously and to "guff" (deliberately leaking round mouthpiece) if necessary.

Hypoxia: Nitrogen accumulation in the respiratory circuit with resultant hypoxia was inevitably considered in view of the shallow depth and the severe subjective disturbances. The almost "compulsive" repeated washing out of the lungs and apparatus before diving on oxygen, which was then practised in the Royal Navy, rendered this most unlikely. Other possible sources of nitrogen such as tissue nitrogen being excreted during the dive or nitrogen in the cylinder gas were carefully assessed and it was concluded that the risk of hypoxia was extremely remote even in the most unfavourable circumstances.

Hyperoxia: The syndrome was totally unlike oxygen poisoning (no convulsive symptoms, no lip or face twitching, rapid recovery even after losing consciousness). The Admiralty Experimental Diving Unit was unable to demonstrate oxygen poisoning in dives in the range of 0 to 20 fsw. Signs and symptoms of acute oxygen poisoning were elicited, however, in oxygen divers working hard at 25 fsw. (Donald, Appendix (ii), RNPRC 1944/125).

Hypocarbia: Hyperventilation due to various degrees of anxiety can occasionally occur in diving novices who are not exercising. It is exceedingly difficult to lower the body PCO_2 by hyperventilation at all markedly in breathing apparatus while under pressure. In any case this was not relevant to the shallow water black-out (SWBO) which was occurring in well trained and selected oxygen divers while swimming.

Hypercarbia: It was here that Barlow and MacIntosh (RNPRC, 1944/125) made a remarkable breakthrough which is not easy to appreciate nearly fifty years later. To most physiologists increased inspired CO_2 meant increased ventilation and they would have agreed with Samson Wright that "Poisoning with CO_2 can never be inadvertently, because of the choking sensation aroused" (Applied Physiology, 7th ed., 1942). Yet they felt that possible inadequate CO_2 absorption and increased dead space could cause a rise of CO_2 in the circuit especially during exertion. They became aware that there appeared to be no proper study of the effects of high concentrations of CO_2 in the absence of oxygen lack in human subjects. They therefore exposed subjects to increased CO_2 levels while breathing oxygen by three different methods.

(a) By rebreathing 50 litres of oxygen without CO_2 absorption while at rest, eventually causing signs and symptoms very similar to those of SWBO but "without any notable respiratory distress".

(b) By exercising on a bicycle ergometer while breathing oxygen through a large external dead space (800 ml). Most subjects suffered various disturbances of consciousness and two subjects actually lost consciousness after about three minutes.

(c) By breathing 5% 1 AT CO_2 in oxygen. Some subjects complained of mild symptoms such as slight vertigo and "tingling of the limbs".

The signs and symptoms, particularly in procedure (b), completely matched those encountered in the Shallow Water Black Out (see above). Barlow & MacIntosh also emphasised that some subjects had little warning of its onset in the form of respiratory distress. They proposed, without direct evidence, that "the primary cause would appear to be the failure of the canister to absorb adequately the large amounts of CO_2 produced during muscular work."

During this period the Admiralty Experimental Diving Unit had been carrying out a large series of investigations to determine accurately the oxygen uptake of booted and fin swimming divers in all degrees of operational activity (Donald & Davidson, 1944). Another critical piece of information emerged. It was shown that finned underwater swimmers were capable of much higher oxygen uptakes than the booted divers. These uptakes were of a far greater order (2.5 to 4.0 1/min) than ever encountered or indeed expected, in any underwater activities. The original carbon dioxide absorbent canister used by oxygen breathing frogmen were certainly not designed for such levels of oxygen uptake and of carbon dioxide production. Thus Barlow and MacIntoshs' suspicions were amply confirmed.

The inadequate canister first used by frogmen had been designed and produced outside the usual orbit of manufacture and testing. No other operational breathing apparatus showed this defect nor did any other type of diver reach such high levels of CO_2 production. Nevertheless, even more attention was now being paid to the efficiency of CO_2 absorption with careful determination of the maximal operational demands. Not

only were canisters increased in size but their uniform and proper filling without "channeling" was assured by continuous shaking or by automatic "hopper" filling techniques. The occurrence of channeling (over-used zones of unduly low resistance) in a particular canister model could be diagnosed by using indicator dyes in the absorbent. The effect of water temperatures on the efficiency of CO_2 absorption was studied in the full operational range. Bench testing with air pumps (Kenometer) imitating different patterns and volumes of respiration with the controlled addition of carbon dioxide to the circuit was also developed (Barlow and McIntosh, 1944 (ii))

From 1943, fast or very fast (spurt) swimming by oxygen breathing frogmen was forbidden in the Royal Navy unless demanded by "enemy contact" or other operational needs.

Barlow and MacIntosh did not report individual experimental exposures nor the levels of carbon dioxide achieved when various degrees of intoxication occurred. For this reason the author (Donald, 1945), carried out a series of hyperoxic hypercarbic studies using a fourth method. 18 normal subjects (divers, doctors, visitors) performed moderately hard work on a bicycle ergometer to the rhythm of a metronome while rebreathing pure oxygen from a 50 litre Douglas bag without any absorption of CO_2. The rate of intoxication could, if necessary, be changed by changing the degree of exertion or the capacity of the Douglas bag. All subjects in this particular series exercised at the same rate (VO_2 1.5 1/min. approx.). Gas samples were taken from the Douglas bag at the "end-point".

This simple experiment allowed the assessment of the level of body PCO_2 as the symptoms occurred during exercise and increasing hyperoxic hypercarbia. The measurement of bag PCO_2 at the end-point gave a precise figure. These end-points were between 200 and 380 seconds from commencement and PCO_2 and time, when plotted, were linearly related indicating that equilibrium between lung and bag gases had been achieved, as would be expected with the high production of carbon dioxide and vigorous rebreathing. As premonitory symptoms also occurred in this period and the rate of exercise was strictly controlled, the approximate level of PCO_2 when these symptoms occurred could also be determined.

In this series of 18 exposures only two subjects had to discontinue the experiment because of urgent respiratory distress. Although their inspired PCO_2 was of the order of 60 mm Hg, one had no nervous symptoms and the

other only mild dissociation. The only other subject with severe respiratory distress was stopped by the attendant when acute carbon dioxide intoxication supervened (PCO_2 75 mm Hg). In the remaining fifteen subjects termination of the exposure, usually initiated by the attendant, was due to acute nervous symptoms or signs i.e. severe tremors with loss of balance, dissociation or unconsciousness. Eight subjects had premonitory signs which were fairly consistent i.e. tremor, localised or generalised, "jitterness" and "shakiness". These occurred between PCO_2 levels of 54 to 63 mm Hg in seven of these eight subjects. The acute nervous end-point supervened at 10 to 25 mm Hg higher than this (final PCO_2 69 to 78 mm Hg). The remaining subject of this group, an extraordinarily tough and motivated Diving Chief Petty Officer, had marked arm tremors at a PCO_2 of 58 mm Hg. He continued to exercise for another two minutes. He was now suffering severe generalised tremors and was so highly dissociated that he kept on pedalling 20 seconds after being turned on to air breathing. His final PCO_2 was 98 mm Hg (14% 1 AT).

The remaining eight subjects without premonitory signs had acute CNS intoxication at PCO_2 levels of 50 to 78 mm Hg. The most sensitive subject (50 mm Hg), the only one below 57 mm Hg, suffered twitching and dissociation. The mean end-point of all subjects was 71 mm Hg. (for further details see Donald, 1945).

Ventilatory volumes were not measured in these experiments but it appeared that, in most cases, they were of a high order. It is an intriguing possibility that, in some subjects, the increasing carbon dioxide intoxication may have dulled or even suppressed awareness of respiratory discomfort (dyspnoea). Looking back at this work (1944) it would have been of great interest to repeat these experiments using a box-bag spirometer (Donald & Christie, 1948), as not only ventilation but the balance of gas exchange between subject and circuit could have been observed directly.

Finally it is of interest to note that Paul Bert (1878) had already clearly demonstrated the auto-intoxication of animals by their own carbon dioxide when in super-oxygenated closed spaces. He noted the remarkable tolerance of mammals to transient, severe hypercarbia and suggested that carbon dioxide should be seriously considered as an anaesthetic during surgical procedures. He proposed the administration of a 40% CO_2, 60% O_2 mixture. However self-contained oxygen rebreathing apparatus, although

suggested by several workers and developed by Fleuss (1876), had not been established and Bert had at no time considered hyperoxic hypercarbia as a potential danger to the diver.

After the CO_2 absorption defect in the diver's breathing apparatus had been remedied and fast swimming avoided, Shallow Water Black Out, as described, largely disappeared. Nevertheless unexpected impairment or loss of consciousness underwater or while surfacing was, and still is, encountered due to a host of causes such as oxygen poisoning; carbon dioxide intoxication; hypoxia due to inadequate purging and oxygen washout (rare); hypoxia due to incorrect use of oxygen-nitrogen mixtures, particularly near surface; sudden exacerbation of hypoxia when ascending from over-long breath-hold dive; gas cylinder errors, incorrect gas or inadequate filling; nitrogen narcosis with or without added CO_2 intoxication; cerebral aero-embolism; severe vertigo (usually otobaric, sometimes with severe disorientation); psychological disturbances i.e. phobias, hysteria, etc; "clinical" conditions i.e. epilepsy with variants, acute cardiovascular events, blood sugar and other metabolic disturbances and so on.

Moving on to the next decade, Lambertsen et al (1955) demonstrated that, when human subjects were breathing oxygen at 3.5 ATA (in the dry), the administration of 2% (1 AT) carbon dioxide caused the jugular venous PO_2 to rise dramatically from 100 to 1000 mm Hg. This finding further supported the view that the effect of carbon dioxide on oxygen toxicity was due to cerebral vasodilatation and increased cerebral blood flow. Lambertson also found that exercising (VO_2 2.0 1/min. approx.) at 2 ATA while breathing oxygen, resulted in a lesser ventilation than when exercising on air at 1 AT, but the resultant rise of arterial blood PCO_2 was not significantly above normal levels and cerebral blood flow was unaltered. He concluded that the cause of the increased toxicity with exercise lay elsewhere.

The Carbon Dioxide Retainers

Lanphier (1955, (i)) carried out the first US Navy trials testing the safety of the principle of partial pressure in relation to oxygen-nitrogen mixture (ONM) diving. Somewhat dramatically, in his first experiment a diver breathing 47% ONM at 99 fsw (PIO_2 1.88 ATA, DO_2 29 fsw) convulsed after twenty minutes. In view of this event and a number of lesser incidents (see

Chapter 5 for details), Lanphier reported that "a given partial pressure of oxygen was simply proving more toxic in the presence of increased nitrogen pressure." The present author would not agree with this conclusion (see Chapter 5).

Nevertheless as already noted in Chapter 5, Lanphier raised a number of important points concerning the breathing of oxygen-nitrogen mixture at increased depths. He considered the possible factors that might add further to the dangers of oxygen poisoning in these conditions. Naturally the role of carbon dioxide was carefully examined. In the experiment mentioned above the subject was breathing oxygen-nitrogen mixture from an open circuit. Thus dead space and inspired PCO_2 were strictly controlled. There were other possible reasons why an oxygen-nitrogen mixture diver (say at 99 fsw) might have a reduced ventilation and raised body PCO_2, particularly while exercising i.e. hyperbaric oxygen (1.8 bar), hyperbaric nitrogen (2.2 bar) or more importantly, increased respiratory resistance, both internal and external, due to increased gas density. Lanphier also considered the possibility that there might be certain individuals whose ventilatory response to exercise was consistently less than the accepted normal and that this tendency might be aggravated by such adverse factors. He therefore carried out a large series of studies (1954 to 1958) on teams of trained divers to determine whether there were such "under-ventilators" or "CO_2 retainers" and whether they could be reliably identified and, possibly, excluded from certain types of diving. He made a particular study of ventilatory response and end-tidal PCO_2 during exercise in these dives.

Before describing his experiments and findings, it is worth considering the value of end-tidal PCO_2 measurements as a great deal hangs on this. It is generally agreed that the end-tidal PCO_2 is not an accurate measure of the mean arterial blood PCO_2 during exercise (Dubois et al, 1952; Asmussen & Neilson, 1956; Asmussen, 1965, Jones et al, 1979). The alveolar "CO_2 plateau" rises throughout expiration, particularly with significant exercise and increased tidal volumes. Dubois et al (1952) established that the CO_2 level measured at 60% of expiration during exercise is nearer to the mean arterial blood PCO_2 values. The increase of respiratory pressure gradient at the mask or mouthpiece due to increased resistance may also interfere with the free and uniform flow of sampled gas both in the diver's lungs and in the apparatus. Somewhat unpredictable hydrostatic

gradients between the swimming diver and his breathing apparatus may also affect gas distribution and sampling. Increased resistance is inclined to slow the diver's respiration and increase the tidal volume.

These considerations also bring us to the question of the diver's respiratory pattern and its effect on end-tidal sampling. At the time of these experiments and for some years after, many divers made a habit of breathing at much slower rates with larger tidal volumes even when exercising. A rapid and deep inspiration is often followed by a considerable pause, in which the glottis may be closed, followed by expiration. This action is not dissimilar to that of a fast surface swimmer snatching and holding air. In the case of the diver it is considered, by some, to be a reaction to respiratory resistance. Others consider it to be an acquired habit of breathing. There is no doubt that many divers were trained to breath in this way as it was thought to economise in the amount of gas demanded. It is likely that "skip" breathing, as it is called, with its element of transient breath holding, would certainly affect end-tidal sampling, as would abnormally slow breathing alone. Divers are now trained to avoid skip or very slow breathing (also called "controlled" breathing).

Lanphier's investigations with a whole variety of depths and inspired gases are far too extensive to review in detail. Yet as his exceedingly well documented original accounts have never been published, it is worth looking at the major features. These experiments were carried out in a "wet" pressure chamber with the "finned" diver swimming underwater against a trapeze (8 lb. thrust). The mean oxygen uptake was 1.4 1/min. The diver was on an open circuit with a mask and fitted mouthpiece. The appropriate gas was supplied through a demand valve. Total respiratory pressure swings at the mouth were also recorded in a number of dives. The expired gas PCO_2 was monitored throughout. The end tidal PCO_2 reported was the mean of the end-tidal values during the last five minutes of fifteen minutes exercise. All end-tidal PCO_2 figures in the following text are the means of each group during exercise unless specified otherwise. In view of other studies of subjects exercising while wearing respiratory apparatus (Lally et al, 1974, Broussolle et al, 1972, Piantadosi, 1974, Cotes, 1965) end-tidal PCO_2 levels at or below 48 mm Hg have been quite arbitrarily considered "unremarkable" by the present author. Individual end-tidal PCO_2 figures for all dives are given separately in Table 29,

column 7 (above 48 mm Hg) and column 8 (48 mm Hg and below). The order of presentation is the order of the exposures and has no other significance.

In the *first series* (Phase I & II) Lanphier (1955, (ii)) wished to compare the ventilation and alveolar PCO_2 (end-tidal) when breathing oxygen at 26 fsw and 40% ONM at 99 fsw (DO_2 26 fsw). He was not studying oxygen poisoning under these conditions and he shortened all exposures to 15 minutes. He was essentially interested in the effects of increased density of gas when oxygen-nitrogen mixtures were used at 99 fsw. Seventeen subjects were involved in all the exposures. A proving run on air near the surface was first completed, then the dives to 26 fsw on oxygen and to 99 fsw on 45% oxygen-nitrogen mixture.

As will be seen in Table 29, the mean measured end-tidal PCO_2 was significantly higher in the 45% oxygen-nitrogen mixture dives at 99 fsw (53.5 mm Hg), than in the pure oxygen dives at 26 fsw (46.1 mm Hg). Out of the seventeen divers on the oxygen-nitrogen mixture at 99 fsw, thirteen had end-tidal PCO_2 levels above 48 mm Hg. The values ranged from 50 to 63 mm Hg.

It will also be noted in Table 29 that six divers on oxygen at 26 fsw and five divers on air at 4 fsw showed abnormally raised exercising end-tidal PCO_2 levels (above 48 mm Hg) and that the mean end-tidal PCO_2 was 46.1 and 46.7 mm Hg respectively.

Although the mean ventilatory volumes were slightly less on oxygen-nitrogen mixture at 99 fsw than on oxygen at 26 fsw this was in accord with most dives at 99 fsw. In any case the difference was not significant.

Next the same group dived to 99 fsw breathing 45% oxyhelium mixture (OHeM). Approximate calculations would indicate that the lower density of the OHeM would result in the internal and external respiratory resistance at 99 fsw being of the order of that on air or oxygen at 20 fsw. A considerably lower mean end-tidal PCO_2 (46.9 mm Hg) was observed on the oxyhelium mixture than on the oxynitrogen mixture (53.5 mm Hg). The exercising ventilation in the two studies was, surprisingly, almost the same (ONM 24, OHeM 24.5 1/min.). Again, nine out of the seventeen divers breathing the helium mixture at 99 fsw still showed raised (above 48 mm Hg) exercising end-tidal PCO_2 tensions (see Table 29). This observation, as well as the findings on oxygen at 26 fsw and on air at 4 fsw, suggests there are other important factors apart from gas density, affecting the end-tidal PCO_2 levels as measured.

TABLE 29

VENTILATION AND END-TIDAL PCO_2 (GROUP MEANS) IN EXERCISING DIVERS AT VARIOUS DEPTHS AND BREATHING VARIOUS GAS MIXTURES. (Data from Lanphier, 1955 (i) & Lanphier, 1958)

	Depth fsw	Gas Breathed	Number of Subjects	Mean End-Tidal PCO_2 mm Hg	Mean Respiratory Minute Volume 1/min.	No End Tidal PCO_2 > 48 mm Hg	Individual Subjects with End-Tidal PCO_2 > 48 mm Hg	Individual Subjects with End-Tidal PCO_2 < 49 mm Hg
SERIES I	4	Air	17	46.7	27.2	5	55,51,54,51,52	43,48,48,38,48,44,42,39,46, 44,44,46
	26	Oxygen	17	46.1	26.7	6	56,53,49,51,51,50	42,48,47,34,46,38,40,47,39, 45,47
	99	45,ONM	17	53.5	24.0	13	51,60,56,54,63,61,61,58,50, 59,56,52,53	40,40,47,47
	99	45, OHeM	17	46.9	24.5	9	57,49,51,54,49,55,50,54,49	45,32,41,39,43,44,43,43
SERIES II	4	Air	10	47.1	28.2	4	49,51,54,49	40,32,41,39,43,44,43,43
	4	Oxygen	10	48.3	25.6	6	49,49,49,55,49,56	37,46,48,44
	99	45,ONM	10	51.3	22.6	7	55,70,55,51,50,53,49	47,43,40
	99	45,ONM	10	50.6	20.9	7	50,60,50,56,49,64,55	48,47,46,41,45,38,44
	99	Air	10	49.4	20.9	5	50,61,55,49,56	46,46,38,47,45
	99	Air	16	49.0	21.8	8	50,58,53,60,53,49,50,58	47,46,47,46,47,40,40,44
	99	Air	13	49.8	20.7	8	53,51,56,62,51,54,52,52	40,48,43,42,43
	99	5-7, ONM	10	48.6	23,3	4	53,55,53,49	45,45,48,48,41,47
	99	45,OHeM	10	44.6	25.0	2	53,51	45,47,48,38,43,37,42,42

The diver CLL (Lanphier, 1955, (i)) who convulsed after 20 minutes exercise on 47% ONM at 99 fsw (DO_2 29 fsw) had the following findings in this first series:

GAS	DEPTH (FSW)	RESP. MIN.VOL. (1/min)	$PETCO_2$ (mm Hg)
Air	4	21	55
Oxygen	26	21	56
45% ONM	99	20	59
45% OHeM	99	23	57

His ventilation and end-tidal PCO_2 during standard exercise appeared almost independent of the gases breathed or of the depth of the exposure.

In this first series there were five out of the seventeen divers who, like CLL, showed a "raised" end-tidal PCO_2 (above 48 mm Hg) in all the four exposures at different depths and with different gases. It is worth examining their respiratory behaviour during these exposures.

In three of these divers the mean exercising respiratory rate in all four exposures was 7 per minute, consequently the tidal volume was considerably increased (3.5 1) compared with the mean group value (2.4 1). The respiratory rate in each diver was almost constant and not related to the density of gas breathed. In the other two subjects of this "hypercarbic" group the respiratory rate was slightly higher, although still below the group means, and the respiratory minute volume was persistently lower (about 20% less than the group means). Thus tidal volumes were near to the group averages. It is difficult to be sure whether the breathing of these divers is controlled reflexly or is strongly influenced by higher centres. The almost fixed degree of bradypnoea under such varied conditions suggests an acquired rather than a reflex pattern of breathing.

In the *second series Phase 4 & 6* (Lanphier, 1958) it was shown that the mean end-tidal PCO_2 of the group, while exercising at 99 fsw, was not significantly different when breathing 45% ONM (two studies, $PETCO_2$, 51,51), air (three studies, $PETCO_2$, 49, 49, 50), or 6% oxygen in nitrogen (one study, $PETCO_2$, 49 mm Hg). The mean exercising respiratory volume was between 21 and 23 1/min. in all cases (see Table 28). These findings would support the proposition that the density of the gas breathed is an important factor affecting the level of the sampled end-tidal PCO_2. As oxygen and nitrogen have roughly similar densities (13.9 N_2: 15.90$_2$) the

proportions breathed will not be critical in this regard. It also appears that the effect of varying tensions of oxygen and nitrogen on the level of the exercising ventilation at these pressures is not of great importance.

The 45% OHeM dives to 99 fsw in this second series showed a mean exercising end-tidal PCO_2 of 45 mm Hg. Only two out of the ten subjects had an end-tidal PCO_2 above 48 mm Hg (53, 51). The ventilation was about 3 1/min. greater than that found in the ONM dives in this second series.

There was no oxygen study at 26 fsw in this series but one at 4 fsw, on oxygen, which showed abnormal end-tidal PCO_2 on exercise (over 48 mm Hg) in six out of ten divers. Such findings and, indeed, many of those shown in Table 29, raise considerable doubts as to the significance of the end-tidal PCO_2 figures.

Ventilation Although the exercise ventilation at 99 fsw was in general a few litres per minute below that at or near surface, the level of exercise ventilation did not correlate reliably with the level of the end-tidal PCO_2 levels either in groups or in individuals.

Respiratory Rhythm The mean respiratory rate of the divers at all pressures (4 to 99 fsw) was about 0.6 of the predicted rate at that level of exercise ventilation.The divers with the slowest breathing almost always had the highest end-tidal PCO_2 levels.

Resistance The total respiratory pressure swing at the mouth during these dives varied between 29 and 18 cm H_2O, the inspiratory negative pressure being slightly greater than the expiratory positive pressure. This resistance is certainly greater than that encountered in most operational work, particularly when there are no valves as in pendulum breathing.

Hypercarbia One surprising feature was the almost total lack of signs and symptoms suggesting a true hypercarbia of the degree indicated by the end-tidal PCO_2 measurements. Again, despite the apparent hypercarbia in many instances, there were no signs or symptoms of oxygen poisoning while exercising at an oxygen partial pressure of 1.8 ATA for 15 minutes and, in some cases, for 30 minutes. (15 min. ONM + 15 min. OHeM).

Controls It should be emphasized that the level of the exercising end-tidal PCO_2 was well above 40 mm Hg in a very large majority of divers at all depths (4 to 99 fsw), no matter what gas was being breathed (see columns 7 and 8, Table 29). The high mean end-tidal PCO_2 in all groups confirms this. It was obviously not possible to repeat these exercising dives

with non-diving subjects in these wet chamber conditions. However there must be a considerable suspicion that any subject, whether a diver or not, might well show a moderately "raised" pressure performing the same level of exercise and using the same respiratory circuit and gases. Another possible approach to some sort of control would have been ventilatory and end-tidal PCO_2 studies of the same diving trainees before and after minimal instruction, after full training and finally, after significant operational experience.

Test of End-Tidal PCO_2 Sampling Technique

For these and other reasons mentioned earlier, Lanphier was anxious to check the validity of his end-tidal sampling technique against arterial blood PCO_2 measurements, particularly during exercise.

This was carried out in the Department of Physiology in the University of Pennsylvania between the first and second series of the dives (Lanphier, 1956, (i)). The whole study was performed while breathing air at atmospheric pressure in the laboratory. Five of the six subjects were divers who had shown some of the highest end-tidal PCO_2 levels in the working dives to 99 fsw breathing 45% ONM (60, 56, 61, 61, 58 mm Hg). The sixth subject was the investigator who had never shown a raised exercising end-tidal PCO_2 in many dives. The respiratory circuit and end-tidal sampler were the same as those used in the US Navy Experimental Diving Unit underwater studies except that a "hydrostatic resistor" was attached to the expiratory side to "simulate" underwater conditions. The mean pressure swing at the mouth during exercise was -12.8 to +13.2 cm H_2O. The subjects exercised on a bicycle ergometer at the usual level (mean VO_2 1.4 1/min.), in the supine position. Arterial blood and end-tidal samples were drawn simultaneously over the whole of the last five minutes of fifteen minutes rest (supine) and of fifteen minutes exercise.

There were thus twelve simultaneous blood and gas samples to compare. The end-tidal PCO_2 differed from the arterial blood PCO_2 by 3 or more mm Hg in seven instances. Nevertheless the matching was, on the whole, fairly good (See Table 30). The resting end-tidal PCO_2 was consistently lower than the arterial blood PCO_2 (mean -3 mm Hg), and the exercising end-tidal PCO_2 was higher in four instances (mean +2 mm Hg). Although Lanphier appeared satisfied with this comparison in so few

subjects at atmospheric pressure, a much larger series is desirable under different conditions particularly at increased pressure.

With regard to the carbon dioxide levels during exercise (see Table 30), Lanphier concluded that, even on air at one atmosphere, the "CO_2 regulation" of these divers was different "to the norm" and that "characteristics inherent in the diver – subjects are responsible".

TABLE 30

REST AND EXERCISE ARTERIAL BLOOD AND END-TIDAL PCO_2 IN MIXTURE DIVERS BREATHING AIR AT ONE ATMOSPHERE. (FROM DATA OF LANPHIER, 1956)

1 & 2 Simultaneous samples using Expl. Diving Unit circuit and end-tidal sampler (EDU)
4. End-tidal sampling only, using Lambertsen's low resistance circuit & end-tidal sampler (l)

State	Rest						Exercise (VO_2 1.4 1/min					
Subject	CLL	CO	F	H	HL	L	CLL	CO	F	H	HL	L
1. Art. Blood PCO_2	*(49.5)	44	39	34	44	37	(57)	47	44	44	51	35
2. End Tidl (EDU) PCO_2	(45)	41	35	32	40	37	(53)	49	46	47	50	38
3. ΔPCO_2 mm Hg	(-4.5)	-3	-4	-2	-4	0	(-4)	+2	+2	+3	-1	+3
4. End Tidl. (l) PCO_2	(42)	43	•29	43	40	38	(51)	46	41	48	44	38

* Note abnormal resting arterial blood PCO_2. See text.
• Hyperventilated

The present author would not accept these conclusions. Firstly, one subject CLL, had a *resting* arterial blood PCO_2 of 49.5 mm Hg, which, no matter what the reason (obstructive respiratory disease, medication, error) made him "unacceptable" in this study. Excluding CLL and the investigator (L) in the first exercise study, only one of the four divers showed a raised (above 48 mm Hg) arterial blood PCO_2 (51) on exercise and two a raised end-tidal PCO_2 on exercise (49, 50). However the resistance of the circuit, of which several subjects complained, was considerable. The respiratory pressure at the mouth during exercise was – 12.8 to +13.2 cm H_2O (mean).

For this reason alone, control experiments with non-diving normal subjects using the same procedure, circuit and techniques, would appear to be essential before comparison is made with the "norm".

In the second rest-exercise study (see Table 30) the same subjects breathed from a Lambertsen low resistance, low dead space circuit. The pressure swing during exercise at the mouth was – 1.5 to 2.7 cm H_2O. Only end-tidal samples (Lambertsen sampler) were taken. Excluding CLL, none of the four divers raised their end-tidal PCO_2 on exercise to above 48 mm Hg, the mean figure being 44. Again, it would have been of great interest to have had a control group of non-diving normal subjects.

Identification of "Carbon Dioxide Retainers"

In the diving studies described certain individuals had show markedly raised end-tidal PCO_2 levels fairly consistently, not only on 45% ONM, but also when breathing air, oxygen and other oxygen-nitrogen mixtures. Lanphier had hoped to identify a definite group of "carbon dioxide retainers" who might be at particular risk during oxygen-nitrogen mixture diving. However he was unable to "define a discrete group of individuals especially subject to this effect."

Initially he had considered the possibility that the CO_2 "retainers" would show reduced ventilatory response to exercise at pressure (say 99 fsw). Although, in general, the exercise ventilation was almost always a few litres (per min.) below that at surface, no matter which oxygen-nitrogen mixture was being breathed, in individual instances the level of ventilation did not correlate reliably with the level of end-tidal PCO_2.

In looking for a test to identify "carbon dioxide retainers" it was logical to test the ventilatory response of divers to increased inspired tensions of carbon dioxide. In the Philadelphia study just described (Lanphier, 1956, (i)), the same five divers who had been specially selected because of their high end-tidal PCO_2 levels when breathing 45% ONM at 99 fsw, were also tested for their ventilatory response to various levels of inspired CO_2 in air (3.9, 5.0 and 6.6% 1 AT).

Lambertsen's low resistance, minimal dead space circuit and end-tidal sampler were used. The results are shown in Table 31 (mean group values only). The figures given in the table for "normal non-divers" are derived from large studies in the same laboratory, using the same apparatus and

procedure (Lanphier, 1956). As a group and individually, the ventilatory response of the divers to inspired CO_2 was entirely normal. It is not generally appreciated that these five divers, who could be fairly described as the original "carbon dioxide retainers", had a normal ventilatory response to inspired carbon dioxide.

TABLE 31

VENTILATORY RESPONSE TO INCREASED INSPIRED CO_2 IN AIR IN FIVE MIXTURE DIVERS (SEE TEXT) AND NORMAL SUBJECTS

SUBJECTS		Air	3.9% CO_2 IN AIR	5.0% CO_2	6.0-6.6% CO_2 IN AIR
Normal	Mean End-tidal PCO_2 (mm Hg)	39	43.3	46.2	47.5 (6%)
(Non-Divers)	Mean Respiratory Min. Vol. (1/min) BTPS	6.0	16.0	25.5	30.8 (6%)
Divers (see text)	Mean End-tidal PCO2 (mm Hg)	35	43	46	51.2 (6.6%)
	Mean respiratory Min. Vol. (1/min) BTPS	8.7	15.9	22.0	35.4 (6.6%)
	Range of individual divers respiratory min. vol. (1/min)	6-10	12-19	18-27	28-47

All the divers responses are well within the accepted normal range throughout. Normal values (means only) derived from studies in Pharmacology Dept. Range not available. Data derived from Lanphier, 1956.

Finally Lanphier (1956, (ii)) studied the effect of added dead space (1 litre) while exercising on a bicycle ergometer (VO_2 1.4 1/min. approx.) and breathing air at 1 and 4 ATA. The subjects were the same five divers of the Philadelphia study, except CLL, whose place was taken by another diver who had also shown a high end-tidal PCO_2 (63 mm Hg), at 99 fsw on 45% oxygen-nitrogen mixture (Lanphier, 1956). At atmospheric pressure, of the five divers, the end-tidal PCO_2 fell with the added dead space in three subjects and rose in the other two. At 4 ATA the added dead space caused

the end-tidal PCO_2 to rise in three subjects, fall in one and remain unaltered in another. Again the level of end-tidal PCO_2, even with one litre dead space and exercise on air at 99 fsw, did not approach those achieved by the same divers when breathing 45% oxygen-nitrogen mixture at 99 fsw with minimal dead space. The dead space used here was a collapsible rubber bag on the proximal expiratory circuit which first emptied on inspiration before the demand valve was activated. Lanphier was at loss to explain these strange findings and suggested further experiments using a more orthodox circuit and rigid dead space.

Thus Lanphier was unable to find any relatively simple test to identify "carbon dioxide retainers".

To summarise, the only evidence in this large series of careful experiments to support the possible occurrence of carbon dioxide retention in exercising mixture divers at depth was the raised end-tidal PCO_2 in the majority of 45 ONM divers at 99 fsw. (See Table 28).

However elevated end-tidal carbon dioxide levels were also found in a considerable number of divers breathing air and other ONM at these depths, of divers breathing oxygen (25 and 4 fsw) and air (4 fsw) at shallower depths, and even while breathing air at atmospheric pressure. Comment has already been made on the lack of control experiments with non-diving subjects in compressed air at 4 ATA and at atmospheric pressure.

As the end-tidal PCO_2 is admittedly an unreliable measure of arterial blood and body PCO_2, particularly under diving conditions, the hypothesis that oxygen-nitrogen mixture divers are at considerably increased risk of oxygen poisoning, due to carbon dioxide retention, remains unproven. Studies by other workers of the ventilatory response of divers to exercise and raised tensions of inspired carbon dioxide are reviewed in some detail in the final chapter.

REFERENCES

ASMUSSEN, E. (1965): Muscular exercise. *Handbook of Physiology, Section 3*, 2, 939-978.

ASMUSSEN, E. and NEILSON, M. (1956): Physiological dead space and alveolar gas pressures at rest and during muscular exercise. *Acta. Physiol., Scandinav.*, 38, 2-21.

BARLOW, H.B. and MACINTOSH, F.C. (1944, (ii)): Measurement of dead space of breathing sets. *Royal Naval Personnel Committee* 44/100.

BARLOW, H.B. and MACINTOSH, F.C. Appendix DONALD, K.W. (1944): U.P.S. 48, *Royal Naval Personnel Committee* 44/125.

COTES, J.E. (1965): Lung function. *Blackwell Scientific Publications, Oxford.*

DONALD, K.W. (1945): Effect of oxygen at shallow depths. *Cambridge M.D. Thesis, Section* 15, 146-154.

DONALD, K.W. and CHRISTIE, R.V. (1948): A new method of clinical spirometry. *Clin. Sci.*, 8, 21-32.

DONALD, K.W. and DAVIDSON, W.M. (1944): Oxygen uptake of divers. *Admiralty Experimental Diving Unit (R.N) Report XV/44.*

DUBOIS, A.B., BRITT, A.G. and FENN, W.O. (1952): Alveolar carbon dioxide during the respiratory cycle. *J. Appl. Physiol.*, 4, 535-548.

FORBES, H.S. (1928): Arch. *Neurol Psych.*, 19, 751.

GIBBS, E.L., GIBBS, F.A. and LENNOX, W. (1935): Regulation of cerebral carbon dioxide. *Am. J. Physiol.*, 111, 557-566.

HILL, L. (1933): Carbon dioxide and oxygen poisoning. *Q. J. Exp. Physiol.*, 23, 49-56.

JONES, N. L., CAMPBELL, E.J.M., EDWARDS, R.H.T. and WILKOFF, W.G. (1969): Alveolar to blood PCO_2 difference during rebreathing in exercise. *J. Appl. Physiol.*, 27, 356-364.

LAMBERTSON, C.J. (1955): Respiratory and circulatory action of high oxygen pressure. *Proc. Underwater Physiol. Symposium, Pubn.* 377, *Nat. Ac .Sc. & Nat. Res .C, Washington D.C.*

LANPHIER, E.H. (1955, (i)): Use of nitrogen-oxygen mixtures in diving. *Proc. Underwater Physiol. Symposium Pubn. 373. Nat. Ac. Sc. & Nat. Res. Council, Washington D.C.*

LANPHIER, E.H. (1955, (ii)). Nitrogen-oxygen mixture physiology. *Phases 1 and 2. U.S. Navy Experimental Diving Unit, Washington, Report* 7-55.

LANPHIER, E.H. (1956), (i)): Nitrogen-oxygen mixture physiology. *Phase 3. U.S. Navy Experimental Diving Unit, Washington, Report* 2-56.

LANPHIER, E.H. (1956, (ii)): Nitrogen-oxygen mixture physiology. *Phase 5. U.S. Navy Experimental Diving Unit, Washington, Report* 5-56.

LANPHIER, E.H. (1958): Nitrogen-oxygen mixture physiology. *Phases 4 and 6. U.S. Navy Experimental Diving Unit, Washington, Report 7-58.*

LEONARD, C. (1942): Personal communication.

WOLFF, H.G. and LENNOX, W.G. (1930): The effects on pial vessels of variations in O_2 and CO_2 content of the blood. *Arch. Neurol. Psych.*, 23, 1097-1120.

CHAPTER 7

ARE DIVERS REALLY DIFFERENT?

During the sixties and seventies there was a remarkable surge of "near folk lore" about differences of the physiological behaviour in divers. These differences were first proposed in informal discussions and later in more formal symposia and journals. In essence the proposal was that divers developed an insensitivity to raised tensions of carbon dioxide and a low ventilatory response to exercise. It was suggested that this was an "adaptation" to the underwater conditions. One enthusiast declared "the underwater man has arrived!". The obvious advantage of reduced ventilatory volumes on exercise is the reduction of respiratory work, particularly with increased gas density and the added resistance of respiratory apparatus. The disadvantage is that the resultant rise of carbon dioxide tensions would increase the risks of carbon dioxide narcosis, of accelerated oxygen poisoning and of the accentuation of nitrogen narcosis.

Nevertheless it became widely considered that all divers developed this feature to different degrees. Those with the most severe degree were titled "carbon dioxide retainers". Some thought that these changes were "for life". Others claimed that they could be reversed by a short period (three months) of abstinence from diving. One worker reported the purported "adaptation" of reduced exercise ventilation and rise of end-tidal PCO_2, with reduction in respiratory discomfort, in a non-diving subject during his second exposure to work at pressure. It was also suggested that, as with oxygen poisoning, the carbon dioxide sensitivity could vary from day to day in a particular diver. Another worker claimed to have shown impaired ventilatory response to exercise in subjects who had been retired from diving for over ten years. He proposed that either their physiological behaviour had changed permanently or that they were

inherently less responsive to exercise and carbon dioxide. It was even suggested that such people might have been unconsciously "self-selected" as divers.

Although these diffuse hypotheses are highly arguable it is, as always, difficult to counter them except by a disciplined and somewhat laborious examination of the evidence. The author has therefore reviewed and commented on a number of frequently cited "accepted" references, mostly those supporting "the difference" of the diver. This review must be, perforce, a limited one and, no doubt, some important references have been omitted, particularly very recent ones. It is for browsing rather than straight reading. I hope the reader will agree that, in these hurried days, a careful and detailed review of well known references can be a useful and sometimes a surprising exercise.

NON-DIVERS AND DIVERS IN UNDERWATER SWIMMING STUDY

Goff & Bartlett (1957, (ii))

This much quoted work showed that trained oxygen breathing underwater swimmers had elevated exercising end-tidal PCO_2 levels in contrast to untrained swimmers, who had normal levels of exercising end-tidal PCO_2. These authors used a circulating channel of a model testing basin. By varying the speed of water circulation the underwater swimmer could be studied in situ, although swimming at different speeds (0.6, 0.7, 0.8, 0.9 and 1.0 knot). Three or four tests were made at each speed on each individual. The exercise was continued for 20 minutes and final measurements taken when a steady state was achieved. The six trained swimmers (age 28-31) were all graduates of the US Navy UDO TWO Training Programme. The untrained swimmers (number not given) were laboratory personnel (age 29-47).

The trained swimmers showed end-tidal PCO_2 levels as follows (mean values): 0.6 knot, 46 mm Hg. 0.7 knot, 48 mm Hg; 0.8 knot, 50.5 mm Hg; 0.9 knot, 51 mm Hg; 1.0 knot, 52 mm Hg. In contrast, the mean end-tidal PCO_2 of the untrained swimmers was 37 to 38 mm Hg at all these speeds.

The authors reported slow deep breaths in the trained swimmers. Four of these also had prolonged post-inspiratory pauses (skip breathing). A figure was shown in this article in which end-tidal PCO_2 (40 to 65 mm Hg) was inversely related to the oxygen ventilatory equivalent (26 to 14 1/per

litre oxygen uptake). However of the 64 points with end-tidal PCO_2 above 48 mm Hg and ventilatory equivalent below 21 litres per litre oxygen uptake, 53 are the plots of three divers with marked post-inspiratory pauses. The authors themselves question "whether the slow, deep breaths with prolonged inspiratory pauses (and therefore a prolonged CO_2 build-up time) in four of the trained swimmers, would apparently accentuate the already great cyclic variations in the alveolar CO_2 as a result of exercise."

This study, although superficially convincing, has some defects. Quite surprisingly, the number of untrained subjects remains unspecified. Despite the authors concern about the effects of skip breathing, the ventilation, respiratory frequency and tidal volumes are not given. The quite remarkable constancy of mean end-tidal PCO_2 (37.1 to 38.5 mm Hg) at all speeds of swimming in the untrained subjects is not discussed.

Again, exactly what is meant by an untrained subject who can swim underwater? Goff et al (1957, (i)) state, in an associated paper on the work efficiency of underwater swimmers, "these laboratory personnel were thoroughly indoctrinated in the use of closed circuit breathing apparatus but not considered to be well trained or conditioned underwater swimmers since training was conducted only to the point of fairly uniform performance." It is possible that some of the laboratory personnel used in this study, had already taken part after training, in about twenty 20-minute dives in the first work-efficiency study (Goff et al, 1957, (i)). Even if they had not done so, one would have thought that some "training" would be apparent after indoctrination and a roughly similar period of underwater swimming in the present series. In the work efficiency study (1957, (i)), Goff and his colleagues carried out a second series of dives (three to four 15-minute dives at five different speeds). They showed a marked rise of the "untrained" divers' efficiency. There was a fall of oxygen uptake of the order of 300 ml in the range of oxygen uptake of 0.9 to 1.7 1/min. (0.7 to 1.0 knot). However throughout both studies, apparently, the increasing experience and efficiency of these subjects did not lead to any increase of end-tidal PCO_2 during underwater swimming. The authors (see above) appear to be inferring that a considerable period of diving is necessary to become "conditioned". One also assumes that the term "conditioned", as they use it, means either the adoption of "controlled" breathing or the development of genuine carbon dioxide insensitivity (or both) causing raised end-tidal PCO_2 when swimming underwater.

Finally it is important to recall that, at the time of this study (1957), many divers were trained to skip-breath both by instruction and by example. Many considered that this procedure economised in the gas demanded and made the increased respiratory work more tolerable. Yet no mention is made of this salient point when discussing the divers' training or respiratory behaviour. Was skip-breathing known to both groups and was there any discussion of the subject between the groups or with the investigators? Was there any tendency to skip-breathing, or reduced respiratory frequency, as the untrained divers became more experienced?

One would like more data and reassurance that we are not dealing with one group of divers trained or allowed to skip-breath and another group which, with or without instruction, breathed more naturally.

VENTILATORY RESPONSE TO EXERCISE IN DIVERS AT ATMOSPHERIC PRESSURE

Lally, Zechman & Tracy (1974)

This much quoted paper is a most interesting study of the ventilatory responses to exercise in divers (n = 8), "controls" who were non-diving healthy students or laboratory personnel (n = 9) and non-diving runners in training (n = 11). Exercise was performed on a treadmill with 10% gradient at 1, 2 and 3 mph. The divers were the same height as the controls but were three years (mean) older and 12.8 Kg (mean) heavier (119% of control).

The authors related the increment in ventilation on exercise to the oxygen uptake. Both were standardised against weight. All figures discussed are group mean values at a particular speed on the treadmill. They showed that the increase in ventilation per Kg of the divers was less than the "control" value at 1, 2 and 3 mph and significantly less (20%) at 2 and 3 mph ($P \leq .01$). The increase in ventilation per Kg of the divers was not significantly different to that of the runners at 1 and 3 mph, but just significantly lower at 2 mph.

The present author felt that this excellent data could be examined in other ways. The total oxygen uptake and total ventilation were calculated from the various data made available (f, VT, ΔVO_2 /Kg etc.). The oxygen uptake is a reliable measure of the work performed during exercise on the inclined (10%) treadmill. This work is largely a function of the subject's weight. The divers showed a consistently higher oxygen uptake (117-

120%) at each speed than the control subjects, which closely matched their greater mean weight (119% of mean control weight). It was therefore necessary to standardise the oxygen uptake in relation to weight in order to be able to compare the degree of ventilatory response in each group to a *certain level of work.*

However the controls and the divers had almost the same mean height (174.8 and 177.2 cm respectively, a 1% difference). It is difficult to see how the extra weight of the divers would greatly change chest cage size or ventilatory capacity. The situation was as if the divers, of almost the same skeletal size as the controls, carried a very well tailored pack, mostly fat, of 12.8 Kg. For these reasons, the present author considers that the exercise ventilation should *not* be standardised in relation to body weight.

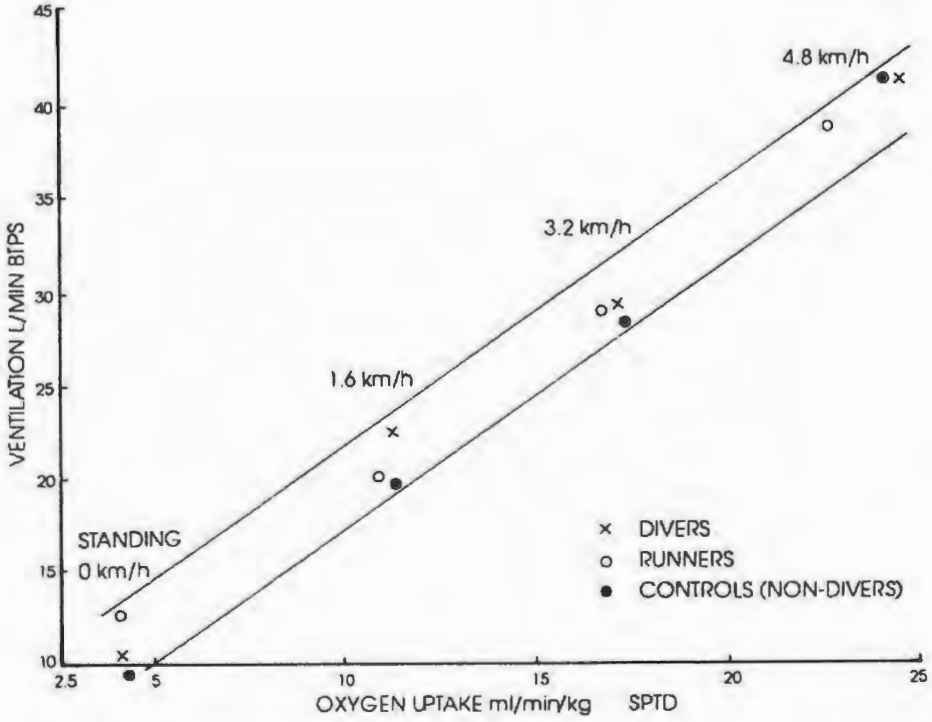

FIG. 23 Ventilatory responses to exercise (treadmill, 10% gradient) in devers, runners and control (see text) ventilation l/lm BTRS V. oxygen/min/Kg. Derived from published data (Lally Et Al, 1974) lines hand-fitted.

In Figure 23 the mean exercise ventilation of the three groups is plotted against the mean standardised (weight) oxygen uptake at 1,2 and 3 mph (10% gradient) on the treadmill. All plots including those of the runners are in a fairly narrow band. The divers' ventilation is almost 3 litres greater than the controls at 1 mph but the ventilation of the two groups become increasingly close at 2 and 3 mph. There is a slight loss of linear relationship at 3 mph. The increased efficiency of the runners (lower oxygen uptake) is also apparent at this speed. It would appear to the present author that the ventilatory response to various degrees of work is almost the same in the three groups.

In this study the expired air PCO_2 was monitored constantly. The PCO_2 at 60% (time) of the expiratory phase was considered a "reasonable approximation" to the alveolar PCO_2 during exercise (Dubois et al, 1952). Using this technique the $PACO_2$ (mm Hg) was between 38 and 39 in all groups when standing before the exercise. At 1, 2 and 3 mph the control subjects mean figures were 42.1, 44.8 and 44.9, the divers 45, 48.4 and 49.6 and the runners 44.7, 47.3 and 47.3 mm Hg. It is of interest that, wearing a "standard SCUBA mouthpiece – valve assembly", the control subjects achieved "alveolar" PCO_2 figures of 44.9 (controls) and 47.3 mm Hg (runners) during high to moderate (VO_2 2.0 to 1.6 1/min.) exertion. Yet some observers would consider these subjects hypercarbic. The divers have a slightly higher figure, presumably because they were doing 20% more work at each stage. Their respiratory rate was lower and the tidal volume about one third greater. Finally, if the end-tidal instead of the 60% expiratory PCO_2 had been measured, the figures would have been even higher.

Kerem, Melamed & Moran (1980)

These investigators studied the respiratory behaviour of large groups of divers (n = 42), ex-divers (n = 58) and "healthy adult males" (n = 49) at rest and during exercise on a bicycle ergometer while breathing oxygen. The subjects were well matched (height, weight, age) except that the ex-divers were ten years older (mean figures). End-tidal PCO_2 and ventilation were measured. There were also breath-holding studies which will not be discussed here. "Each subject was available for one short time only." The subject sat on the ergometer and immediately breathed oxygen ("resting"

state). Measurements were recorded when end-tidal PCO_2, inspiratory flow and pulse rate indicated a steady state (usually after five minutes). The subject then started to pedal. "A steady state gas exchange" was usually achieved after about four minutes of exercise on the bicycle ergometer (650 kpm/min, VO_2 1.6 1/min. approx.). No mention is made of the period of the steady state during which the measurements were made, either at rest or during exercise.

In reporting the results, the authors standardised the respiratory minute volumes for weight, although, unlike Lally's study, the subject's weight did not affect the work done. In any case the mean weight of the three groups were very close (1-1½% difference). Their findings were as follows:

MEAN VENTILATION (1/MIN/KG) BTPS AT REST AND DURING STANDARD EXERCISE

	NON-DIVERS (n = 33)	DIVERS (n = 37)	EX.DIVERS (n = 41)
Rest	0.177	0.141	0.153
Exercise	0.579	0.470	0.490

Both the divers' and ex-divers' mean "resting" and exercise ventilation are significantly different from the non-divers' mean ($P < 0.05$). The authors concluded (abstract) "findings indicated a pronounced hypoventilation and hypercapnia in the divers during exercise."

The reconstructed mean total ventilation figures of each group are more informative and are given in Table 32 with particulars of ventilatory pattern and alveolar PCO_2 (Haldane expiratory sample at rest, end-tidal sample during exercise).

Firstly, as the authors comment, the resting ventilation and "alveolar" PCO_2 figures indicate pronounced hyperventilation which was particularly severe in the non-divers and ex-divers ($PACO_2$ 29 and 32 mm Hg). This was probably due to the unfamiliar environment and experience in the brief "one and only visit" to the laboratory. Such mean resting $PACO_2$ and ventilation findings in groups of this size are, indeed, remarkable. (See Table 32).

With regard to the divers' exercising ventilatory volumes, these are

described as "pronounced hypoventilation". In fact the mean ventilatory volumes of all three groups are within the normal range of ventilation (mean 38, 90% confidence limits 48 and 32 1/min. BTPS) given by Pappenheimer at a VO_2 of 1.6 1/min. working on a bicycle ergometer. Pappenheimer based his figures on material supplied by Dill, Christiansen, Harvard Fatigue Laboratory Data and C. Taylor (The Handbook of Respiratory Data 1944). Nevertheless the difference between the divers' and non-divers' mean exercising ventilation is noteworthy particularly in such large groups. The authors, in considering various factor contributing to or causing this difference, mention the divers' "interrupted' respiration (skip breathing) and their "continued through-a-mouthpiece breathing pattern". They certainly have slower respiration and larger tidal volumes than the non-divers (see Table 32). They also considered the effect of anxiety, manifested by the preliminary hyperventilation, on the levels of exercising ventilation. They found this difficult to assess. Judging by the degree of resting hyperventilation, the non-divers were the most anxious group.

TABLE 32

MEAN TOTAL VENTILATION, VENTILATORY PATTERN AND ALVEOLAR PCO_2 AT REST AND ON EXERCISE

(VO_2 1.6 1/min.approx.)

STATE	RESPIRATORY PARAMETERS	NON-DIVERS	DIVERS	EX.DIVERS
Resting	VE 1/min BTPS	13.17	10.35	11.69
	Respiratory frequency/min.	17	12	11
	Tidal vol. 1 BTPS	0.74	1.01	0.94
	$PACO_2$ (Haldane expy.) mm Hg	29.3	35.6	31.8
Exercise	VE 1/min.BTPS	43.1	34.49	37.44
	Respiratory frequency/min.	20	13	16
	Tidal vol. 1.BTPS	2.15	2.64	2.34
	$PETCO_2$ mm Hg	40.7	48.5	48.0

Data from Kerem et al, 1980

Again, although, in "methods", the authors claimed to have waited for the achievement of a stable pulse rate, ventilatory pattern and end-tidal PCO_2 before commencing and terminating exercise, they state categorically in their discussion that there was no gas exchange steady state during rest. Further they appear to be uncertain about the exercise steady state "although our experience with the system suggested the attainment of a steady state gas exchange after four minutes, even in uninitiated subjects."

Nevertheless the usual resting and exercising periods of 15 minutes would have made a near-steady state more likely and perhaps, would have avoided or reduced the marked hyperventilation in the non-diving normal subjects immediately before the commencement of exercise. It is not unlikely that the most important and critical factor in these studies was the long experience of the divers of breathing circuits in contrast to their "controls".

EXERCISE AT INCREASED PRESSURE (DRY), VENTILATORY & END-TIDAL PCO_2 STUDIES

Jarrett (1960)

This investigator studied four subjects exercising on a bicycle ergometer at 1, 2, 3 and 4 ATA (dry) while breathing air. Each subject worked at three levels (VO_2 0.8, 1.4, and 2.2 1/min. approx.) for 6 minutes with 6 minute rest periods. Three of the subjects were divers and one was a non-diver (laboratory technician) without previous pressure experience.

The three divers all showed raised exercising end-tidal PCO_2 levels particularly at the highest levels of exercise and pressure (64, 65 and 70 mm Hg). End-tidal PCO_2 levels were abnormally raised (above 48 mm Hg) at quite low levels of exercise and pressure. One diver had a raised end-tidal PCO_2 during the three levels of exercise (53, 55 and 58 mm Hg) *at atmospheric pressure*. No diver suffered any respiratory discomfort or fatigue at any time. Only one diver showed a tendency to "controlled" breathing in so far as his respiratory rate was fairly slow (8 to 12) in all states of activity and at all pressures.

The non-diver showed a raised end-tidal PCO_2 (52 mm Hg) during medium exercise (VO_2 1.4 1/min.) at 4 ATA. His ventilation was not

markedly different from that of the three divers at the two lower levels of work. During the highest level of exercise (VO_2 2.2 1/min.) at 3 and 4 ATA he had raised end-tidal PCO_2 levels of 51 and 54 mm Hg, respectively, his ventilation being about 20% greater than the divers' mean. He suffered considerable dyspnoea and exhaustion.

It thus appears that carbon dioxide retention, if judged by end-tidal PCO_2, can occur during exercise at pressure in a non-diving normal subject and that this is not a unique characteristic of divers. When the non-diver repeated the whole experiment (only his second hyperbaric exposure), "he ventilated less, his alveolar PCO_2 rose to higher levels and he was far more comfortable than he had been during his first run." Unfortunately no figures are given. Jarrett claimed priority in describing "the extreme rapidity with which this subject modified his respiratory pattern."

This small but detailed study certainly showed raised end-tidal PCO_2 during exercise at pressure in both divers and a non-diver. However there is no new information as to whether these raised end-tidal PCO_2 levels are truly representative of arterial and body PCO_2 levels in these conditions. There were at least fifteen occasions when the end-tidal PCO_2 during exercise was between 55 and 70 mm Hg. Yet not a single subject appears to have complained of any symptoms of hypercarbia.

Broussolle, Bensimon, Michaud and Vegezzi (1972)

Three experienced naval countermining air divers and three non-diving control subjects were studied at rest and during two levels of exercise (55 & 100 W) at 1, 4, and 7 ATA in a dry pressure chamber. The results are summarised in Table 33, mean group figures being given throughout.

The control subjects showed normal respiratory rates, tidal volumes and ventilation at all pressures and with all degrees of activity. End-tidal PCO_2 levels (continuous sampling, mass spectrometer) were also "normal" although there were readings of 49 mm Hg during the higher levels of exercise at 4 and 7 ATA.

In contrast the divers had remarkably low resting respiratory rates at all pressures (4 to 5 per min.).

TABLE 33

Respiratory data at rest and exercise at 1,4 & 7 ATA

Level of Work	Respiratory Parameters (10th-12th min)	Non-Divers			Divers		
		1 ATA	4ATA	7 ATA	1 AT	4 ATA	7 ATA
0	f	9.4	8.4	11.9	5.0	5.0	4.1
	VTL	1.25	1.1	1.25	1.75	2.05	2.05
	VEL/min	11.7	9.2	14.8	8.7	10.2	8.5
	PETCO2 mm Hg	38	41	42	41	43	44
55W (VO2 1.75 1/min approx)	f	11.7	13.3	11.8	6.2	6.1	4.4
	VTL.	1.5	1.2	1.55	2.3	2.15	3.0
	VEL/min	17.5	16.0	18.3	14.2	13.2	13.3
	PETCO2 mm Hg	41	42	43	45	51	55
110W (VO2 2.4 1/min approx)	f	13.7	13.4	16.8	8.1	6.8	6.3
	VTL.	3.0	2.75	2.2	3.8	3.3	3.5
	VEL/min	41.2	36.95	36.9	30.8	22.3	22.1
	PETCO2 mm Hg	43	49	49	51	61	63

These figures are from published data of Broussolle, Bensimon, Michaud and Vegezzi (1972). Some of the data is derived from diagrams and is approximate.

These rates were only slightly raised on exercise at 1, 4 and 7 ATA (6 to 4 per min at 55 W and 8 to 6 per min at 110 W). The tidal volumes of the divers were therefore considerably greater than those of the controls throughout despite the reduction of the divers' exercising ventilation which was particularly marked at increased pressures. Thus at 110 W the divers' exercise ventilation at 4 & 7 ATA was only 60% of that of the controls. This reduction of exercising ventilation was associated with raised end-tidal PCO_2 levels (51 and 55 mm Hg at 4 and 7 ATA with 55 W exertion and 61 and 63 mm Hg at 4 and 7 ATA with 110 W exertion). The excretion of carbon dioxide was only impaired at the highest level of exertion (VO_2 2.4 1/min. approx.) being 250 ml/min (1 ATA) and 400 ml/min. (4 & 7 ATA) less than that of the controls under the same conditions.

The authors state *"Nous retrouvons la respiration bien caractéristique chez les plongeurs; fréquence basse (environ la moitié de celle des non-plongeurs) qui diminue encore avec la pression; volume courant élevé; pause post-inspiratoire."* Surprisingly they do not identify this as "skip" or "controlled" respiration nor consider whether this marked bradypnoea could be the possible cause of the reduced exercise ventilation and its consequences, at pressure. They demonstrate convincingly that the divers' exercise ventilation was well within the range of their ventilatory capacity. Thus only 40% of the maximal ventilatory capacity was used at 110 W exercise at 7 ATA.

They therefore attributed the reduced ventilation in the divers to their diminished sensitivity to CO_2. Further they emphasised that *"les effets néfastes"* of hypercarbia (CO_2 intoxication and narcosis, increased decompression risks and enhanced nitrogen narcosis and oxygen poisoning) make it necessary to select for *"la plongée profonde avec travail musculaire des sujets qui ne sont pas susceptibles de faire de rétention de CO_2 en milieu hyperbare et qui ont une ventilation maxima élevée."* On these criteria these three senior and highly experienced countermining divers and, presumably, most, if not all, of their colleagues, were unsuitable for diving involving any exertion between 100 and 200 fsw. Again although these divers achieved end-tidal PCO_2 levels between 50 and 63 mm Hg while working at 4 and 7 ATA on air there is no mention of the slightest symptom of hypercarbia or accentuated nitrogen narcosis.

What can explain this difference between these laboratory findings and the reality of diving? It has been suggested that a number of such divers adopt this remarkable slow breathing whenever they are connected to a respiratory circuit in a laboratory. There have been no field studies, as far as I am aware, of the respiratory frequency of such bradypnoeic divers while they are actually on a *real* working dive for significant periods (say 30 to 60 minutes). A strain gauge round the chest and a small recorder would soon be forgotten and would at least determine whether these men have normal respiration during such dives. They should also be studied during their normal activities, including exertion, in everyday life. If these divers' really have a "different" physiology then their respiratory behaviour while breathing air at atmospheric pressure should also be of great interest.

Bradley, Anthonisen, Vorosmarti and Linaweaver (1971)

In this study four experienced and fit divers breathed oxy-helium mixture (30% 1 AT of O_2 throughout) and exercised for 15 minutes on a bicycle ergometer at two levels (VO_2 1.3 and 2.2 1/min. approx.) at sea level, 150, 300, 450 & 600 fsw (dry). 'Alveolar' PCO_2 was calculated (Asmussen and Nielsen, 1956).

The most interesting feature of this study was that two divers had normal respiratory rates, exercise ventilation and 'alveolar' PCO_2 throughout. The other two divers were 'skip breathers' with marked bradypnoea. The ventilatory volumes of the two skip breathers were considerably less at rest and the difference became even greater with increasing exercise and pressure when the minute ventilation was almost half that of the divers with normal respiratory rates. One skip breather (W.L.) had raised 'alveolar' PCO_2 at rest and at all levels of exercise and pressure (up to 61 mm Hg). The other skip breather, surprisingly, only had mildly raised 'alveolar' PCO_2 on two occasions.

TABLE 34

(from data by Bradley et al (1971)

STATE	AMBIENT PRESSURE fsw	SEA LEVEL	150	300	450	600
REST	f per min	4.7	2.3	3.3	3.0	2.7
	VT. L/BTPS	1.6	2.9	2.6	2.0	2.6
EXERCISE	f per min	7.2	3.8	8.2	6.2	7.2
(450 Kg. M/min.)	VT. L/BTPS	3.2	5.4	2.9	3.6	2.9
VO_2 1.2-1.4 1/m						
EXERCISE	f per min	13.2	11.8	11.0	10.6	12.3
(900 Kg. M/min.)	VT. L/BTPS	3.3	3.1	3.4	3.2	3.3
VO_2 2.2-2.3 1/m						

Extreme "controlled breathing" in diver W.L. during rest and exercise.

The respiratory frequency and tidal volumes of diver W.L. are given in Table 34. It will be noted that the resting respiratory rate at pressure is only 2 to 3. Even with moderate exercise (VO_2 1.3 1/min.) the respiratory frequency is between 6 and 8. One reading with moderate exercise at 150 fsw was 3.8 breaths per minute with a tidal volume of 5.4 1.

If the reader tries, with a stop watch, to adopt such rates of breathing, particularly during exercise, he or she will appreciate the degree of abnormality of such respiratory behaviour. In some cases the respiratory cycles were as long as 26 seconds (resting) and 16 seconds (exercising). To achieve such respiratory rates it is necessary to consciously suppress the desire to commence expiration. In other words we are dealing with repetitive breath-holding rather than a natural slow breathing rhythm.

ARTERIAL BLOOD PCO_2 STUDIES DURING EXERCISE AT INCREASED PRESSURE

There are no studies known to the author where end-tidal and arterial blood PCO_2 levels were measured simultaneously during exercise at increased pressures. The only arterial blood studies under such conditions available were those by Salzano and his colleagues (1966 and 1970). In his first study he measured the arterial blood PCO_2 of young, healthy unconditioned subjects carrying out sub-maximal exercise (VO_2 2-2.2 1/min.) on a bicycle ergometer at 1 and 2 ATA, breathing air and oxygen. A Severinghaus CO_2 electrode (gas calibrated) was used. The findings relevant to this review are tabled below (Table 35)

TABLE 35

(Date from Salzano et al, 1966)
Arterial Blood PCO2 During Exercise at 1 & 2 ATA Breathing Air & Oxygen (mean ± SD)

Study	No. of subjects	Gas breathed, ambient pressure. Circuit used	Pre-exercise PCO2 mm Hg	Exercise (8th min) PCO2 mm Hg
1	5	Air, 1 ATA open circuit	33.0 ± 4.9	29.1 ± 9.1
1	5	Oxygen, 1 ATA closed circuit	32.4 ± 5.0	31.4 ± 4.9
2	12	Oxygen, 1 ATA closed circuit	31.6 ± 8.4	39.2 ± 11.5
2	12	Oxygen, 2 ATA closed circuit	32.4 ± 8.3	37.1 ± 7.9
3	8	Oxygen, 2 ATA open circuit	32.9 ± 2.4	43.5 ± 5.4

The mean group resting arterial blood PCO_2 values on air and oxygen at 1 ATA and on oxygen at 2 ATA are extraordinarily low, varying between 31.6 and 33 mm Hg. In the first study (Table 39) the mean exercising level of PCO_2 when breathing oxygen (closed circuit) at 1 ATA was 31.4 + 4.9 mm Hg in contrast to the second study where the mean exercising level breathing oxygen (closed circuit) at 1 ATA was 39.2 + 11.5 mm Hg. The impression gained is that these blood gas estimates (PCO_2) may not be very accurate with a tendency, in some instances, to be too low. This impression is supported by the remarkable finding in this study that in 80 out of 225 instances, during late exercise or early recovery, the PCO_2 of the mixed expired air appeared to exceed that of a simultaneous arterial blood sample (periods of sampling are not specified). As already stated, the end-tidal PCO_2 was unfortunately not recorded in these studies as this may have cast some light on this strange occurrence.

In the second report (Salzano et al, 1970) arterial blood studies were carried out on three subjects (2 divers, aged 27; 1 ex-diver, aged 42) during exercise at three levels (VO_2 1.0, 1.4 and 2.0 1/min.), at one atmosphere (air) and at 31.3 ATA (99.1% He, 0.9% O_2). Each period of exercise was for eight minutes preceded by ten minutes resting seated on the bicycle ergometer. The inspired oxygen at full depth was 220 mm Hg. The oxyhelium mixture at this pressure was 4.4 times denser than air at 1 ATA. At atmospheric pressure no subject's arterial blood PCO_2 exceeded 40 mm Hg during the three levels of exercise. At 1000 fsw the arterial blood PCO_2 during the two lower levels of exercise (VO_2 1.0 & 1.4 1/min.) remained between 40 and 46 mm Hg in all three subjects. However at the sub-maximal level of exercise (VO_2 1.9 - 2.1 1/min.) the two divers raised their arterial blood PCO_2 to 49 and 52 mm Hg respectively. After eight minutes exercise the subjects were taken off the respiratory assembly and breathed ambient gas directly. They continued to exercise for three more minutes. The arterial blood PCO_2 fell to 43 and 47 mm Hg respectively. Although Otis-McKerrow low resistance respiratory valves were used, the external respiratory resistance appeared to be of some significance at this gas density and level of exertion (VO_2 2l/min.). The ex-diver showed normal arterial blood PCO_2 (40 mm Hg or below) even during sub-maximal exercise at full pressure.

Again, it is unfortunate that no end-tidal samples were taken as the previous doubts concerning the accuracy of the arterial blood PCO_2

estimations remain, particularly when one considers the reported arterial blood PCO_2 levels (mm Hg) during rest and exercise in one diver at 1 ATA:

Resting (at least 10 min.) 25: Exercise (VO_2 1.0 1/min.) 31:
Resting (10 min.) 27; Exercise (VO_2 1.4 1/min.) 32;
Resting (10 min.) 26; Exercise (VO_2 1.8 1/min.) 30.

Obviously further hyperbaric studies of arterial blood and end-tidal PCO_2 levels while on circuit at rest and on exercise are desirable.

CARBON DIOXIDE SENSITIVITY IN DIVERS

Schaefer (1955)

Schaefer compared carbon dioxide sensitivity of submarine escape instructors with that of laboratory personnel. These instructors skin-dived, holding their breath, to 90 fsw several times a day for duty periods of up to a year. The ventilatory response was determined while breathing various concentrations of carbon dioxide in air for periods of fifteen minutes. Detailed respiratory data is not given but the results are presented in a figure where mean group values of calculated alveolar ventilation (constant volume of dead space assumed) are plotted against mean group alveolar (end-tidal) PCO_2. The alveolar ventilation is stated in multiples of the values breathing air. The divers' sensitivity to carbon dioxide appears to be less than that of the laboratory personnel. Using the ventilation, breathing air, of the same two groups given in a "hypoxic" study published elsewhere (Schaefer, 1965), we get an approximate idea of the value of S (Δ VE 1/min/Δ $PACO_2$ mm Hg). It is 2.1 for the divers and 2.6 for the controls. The numbers in each group are not given but the author states that the ventilatory response was significantly different, especially in the 5 to 7% CO_2 range (P = .001). It is possible that these results have been published in a more comprehensive form elsewhere.

Schaefer (1965), carried out a further study of seven escape instructors in which he measured the ventilatory response to 5% carbon dioxide in air "during a period of intensive water work" and "after a three month lay-off period". Again the only data presented is a figure showing the mean total ventilation at rest breathing air and that breathing 5% carbon dioxide in air. The minute ventilatory volumes (1/min. BTPS) were as follows:

	Air (1)	5% CO_2 in air (2)	2/1 (Difference n.s.)
Intensive water work	7.3	20.5	2.81
Lay off	8.3	26.0	3.13

The difference between the ventilatory volumes breathing both air and 5% CO_2 in air were significant (P = 0.05).

Finally, it is difficult to see why it was thought that these attendants might adapt to increased carbon dioxide levels. The author showed in a series of tests described in the same paper (1965), that the levels of alveolar PCO_2 (Haldane expiratory samples) were about 25 mm Hg after deep inspiration immediately before the dive, between 40 and 45 mm Hg after the descent to 90 fsw and 40 to 50 mm Hg after an ascent at 1.9 ft/sec. (45.5, SD $\pm$ 3.5). If the ascent is faster (3.5 ft.sec) the alveolar PCO_2 is much lower (31.5 $\pm$ 1.3) due to shortening of the dive and the more rapid expansion and venting of lung gas. Thus, these particular divers only suffer extremely brief periods of very modest hypercarbia.

Froeb (1960)

In this study sixteen professional SCUBA divers were compared with a group of sixteen normal non-diving subjects. Matching of vital statistics was good except that, as is not infrequently the case, the divers' mean weight was 9 Kg (20 lbs) greater than the controls. Froeb found that the absolute values of minute ventilatory responses to 1.6, 3.4 and 5% CO_2 in air, both at rest and during treadmill exercise (VO_2 1.2 - 1.4 1/min.) in the two groups showed no significant difference. However resting minute ventilation on air was different (Divers 7.79; Non-divers 7.16 1/min.) in the two groups, and when minute volume, while breathing carbon dioxide mixtures at rest, was expressed as percentage increase of ventilation on air, there was "significant differences (P = .05)" which the author found "difficult to interpret".

These differences were not found during exercise breathing air and carbon dioxide mixtures (in air). This was because the difference in ventilation during exercise on air in the two groups, was less than in the resting study. Thus, apart from a slight difference of resting ventilation on air, all the other findings at rest and during exercise, while breathing 1.6 to 5% carbon dioxide mixtures, show no significant difference between

divers and non-divers.

Sherman et al (1980) considered that "Froeb's failure to establish a significant difference in the ventilatory response of divers and non-divers could be due to comparison of absolute values of ventilation at relatively low $PICO_2$ where, according to our data, differences are indeed slight." Sherman does not make any such comment about other steady state carbon dioxide studies such as Schaefer (5% CO_2 in one study) or Broussolle (2 to 4% CO_2) which supported the occurrence of reduced CO_2 sensitivity in divers. He also failed to mention Froeb's most impressive demonstration of similar ventilation in non-divers and divers under the combined stimulus of inhaling 5% CO_2 and exercising (2 mph) on a treadmill.

Finally Froeb reported that he found no evidence of slower or deeper breathing than the controls in the divers when breathing air or air and carbon dioxide either at rest or during exercise. This lack of "controlled" breathing may well have contributed to his negative findings. It would have been of great interest to know about these divers' training and whether they had been told not to "skip" breath.

Song, Kang, Kang & Hong (1963)

Song, Kang, Kang & Hong (1963) studied the female breath-holding skin divers (ama) of South Korea. These divers hyperventilate, dive to approximately five metres, collect sea food and then surface after thirty seconds. Each cycle is about one minute and they continue, amazingly, for one hour. Three shifts a day are common. The total time these divers are apnoeic and exercising underwater per day is of the order of ninety minutes. Minute ventilation and alveolar PCO_2 (Haldane expiratory sample) were determined while the subjects were supine and breathing air, 3% CO_2 in pure oxygen and 5% CO_2 in pure oxygen, each for a period of fifteen minutes. Groups of twenty were studied, the controls being housewifes who were well matched apart from being, on average, five years older. The results were as follows, means of groups throughout:

Minute Ventilation 1/min, BTPS and $PACO_2$ mm Hg (Parenthesis)

	Air	3% CO_2	5.5% CO_2
AMA	7.03 (36.0)	12 (40.0)	20 (45.6)
CONTROLS	6.17 (34.6)	12.2 (40.0)	21.6 (44.5)

As will be seen, although ventilation on air is slightly different, the ventilation and alveolar PCO_2 when breathing the carbon dioxide mixtures were remarkably similar. The authors, by expressing the ventilatory response to carbon dioxide in terms of the ventilation on air, purported to show that the ama had a significantly lower CO_2 response ($P<0.05$). A similar exercise with the calculated alveolar ventilation (constant dead space of 100 ml assumed) gave the same significant ($P<0.05$) difference of CO_2 response.

However these are not valid procedures as the air studies are, of course, normoxic and the CO_2 studies are hyperoxic. The data presented here shows only how similar the ventilatory reaction to inspired carbon dioxide in oxygen was in the divers and non-divers. Yet this article is not infrequently cited as an important reference showing that divers are less sensitive to the ventilatory stimulus of carbon dioxide.

These workers also studied the reaction to breathing a hypoxic mixture (8.5% O_2, 91.5% N_2) in the same subjects. The ventilatory response, respiratory rate, tidal volume and alveolar PO_2 and PCO_2 were again remarkably similar in the two groups.

Broussolle, Bensimon, & Onjon (1969)

In this study the ventilatory reaction to 2% and 4% CO_2 in air was observed in eight experienced Naval divers and eight laboratory personnel. Matching was good. The subject breathed air followed by 2 or 4% CO_2 in air for periods of twenty-five minutes. Sampling was carried out during the last five minutes of each state. Each subject carried out four exposures of each carbon dioxide mixture. Only one exposure was carried out on a particular subject in one day. Alveolar end-tidal samples were also collected.

Ventilatory sensitivity to CO_2 (S), stated as Δ 1/min./Δ mm Hg CO_2 was as follows (mean group values):

	Air to 2% CO_2	Air to 4% CO_2
Divers	0.78	1.03
Controls	1.17	1.85

It is unwise to compare 'S' values in different studies with different techniques but these are all very low. Nevertheless the divers show a "significantly" lower sensitivity (P not given). The authors reported very much slower breathing in the divers resulting in the tidal volumes being considerably greater than the controls on 2 and 4% CO_2 (Controls 0.92 & 1.26; Divers 1.47 & 2.18 1, respectively. As the authors comment, there is evidence of some degree of hyperventilation, particularly in the control subjects mean initial end-tidal gas tensions on air, i.e. controls' $PETCO_2$ 34, $PETO_2$ 109 mm Hg; divers' $PETCO_2$ 36, $PETO_2$ 106 mm Hg.

Finally 'B' (non-stimulating level of PCO_2 by extrapolation to zero ventilation) is 31 mm Hg in the controls and 29 mm Hg in the divers. In the absence of any disease these figures can only be caused by hyperventilation.

Florio and Morrison (1979)

In this study the ventilatory response to increased inspired carbon dioxide of ten Royal Navy clearance divers was compared with that of ten non-divers. These control subjects were fit, active laboratory personnel and physiotherapy students. The groups were well matched (age, height, spirometric data) but their weights were not given. The method of Cunningham et al (1957) was used. First the supine subject breathed air for fifteen to twenty minutes until ventilation and end-tidal PCO_2 were stable. The highest level of carbon dioxide (not specified) was delivered to the subject without prior warning. Expiratory volumes and end-tidal PCO_2 and PO_2 were continuously monitored. When these parameters were stable they were recorded for the next five minutes. The process was repeated with at least another four mixtures, each containing less carbon dioxide than the previous mixture. The CO_2 concentrations used were not specified. These exposures were continuous. Stabilisation and measurements generally required about fifteen minutes. End-tidal PO_2 was maintained at approximately 200 Torr regardless of the expired volume or end-tidal PCO_2.

The results were presented in the usual $VE/PETCO_2$ plots. The integrated plots of the individuals of each group show two distinctly separate lines, the divers' ventilation being considerably lower than the non-divers' at all levels of end-tidal PCO_2. The values of 'S'

(1/min/torrCO_2) and 'B' (intercept value, torrCO_2) in the two groups were as follows:

	Divers	Non-Divers	T test (P)
S	2.16 + 0.5	3.25 + 0.99	< 0.05
B	35.3 + 3.4	32.0 + 3.7	< 0.05
VE 1/min. at PCO_2 of 50 torr	32	58	

Thus, as measured in this manner, the mean ventilatory response to carbon dioxide of the divers is "some 33% less than that of the non-divers of similar age and build", 'S' being significantly lower ($P<0.05$) in the divers than the non-divers.

However there are reservations. On first principles the present author is not entirely happy about measuring the sensitivity to the lower levels of inspired carbon dioxide after the subject has been in a continuous hypercarbic state for about an hour. Florio's non-diver $VE/PETCO_2$ plot shows high ventilatory values which are quite abnormally high in the lower range of PCO_2 i.e. VE at 40 torr = 25.5 1/min. When the non-divers plot returned to the original end-tidal PCO_2 on air before the exposure (37.4 torr) the ventilation was very much greater than it had been on air i.e. 17 1/min. in contrast to 9.8 1/min. The inspired PO_2 tension of 200 torr would certainly not account for this marked difference. Thus during these experiments the normal subjects have departed very markedly from their pre-exposure $VE/PETCO_2$ relationship. Again the intercept (VE = O) gives a non-stimulating PCO_2 (B) of 32 torr (Cunningham's normal intercept is of the order of 38 torr).

The divers' plot passes fairly close to the original air VE/PCO_2 point. At 40.3 torr the air breathing VE of the divers was 8.4 1/min. and towards the end of the exposure it was 10 1/min. at this tension.

Thus, although under these experimental conditions the two groups behaved quite differently, it cannot be said that the divers' responsivity is different to the normal as the control plot is, for some reason, undoubtedly abnormal. The levels of ventilation of the control group, particularly in the lower ranges of PCO_2 and the very low value of 'B' (32 torr) suggest that anxiety contributed to the increase in ventilation as well as the induced hypercarbia. We return, as always, to the wisdom of comparative studies of the highly labile function of ventilation in two

groups, when one group has marked and varied experience of breathing from a respiratory circuit and the other group has little or none.

Sherman, Eilander, Shefer & Kerem (1980)

These workers employed the re-breathing method of Read (1967) to study the ventilatory and "occlusion pressure" response to hypercapnoea. The occlusion pressures will not be discussed here except to say that they appear to correlate well with the ventilatory volumes. Twenty normal subjects and twenty-two SCUBA divers were studied. The diving group consisted of sports (7), semi-professional (5) and professional (10) divers, all of whom had more than four years experience. The non-diving control group consisted of university students and medical and laboratory personnel. They were, apparently, "non-athletes". No mention is made of their aquatic habits or experience as subjects breathing from respiratory circuits. The mean weight of the divers was more than 13 lbs greater than the non-divers.

The subject breathed 50/50 oxygen-nitrogen mixture for about five minutes. At the end of a normal expiration he was switched into re-breathing from a bag containing a 7-8% CO_2, 50% O_2 and 42-43% nitrogen. The bag contained about 6 litres (vital capacity + 1-1½ litres) of this mixture. End-tidal PCO_2, breath-by-breath tidal volume and minute volume were recorded. The appearance of the mixed venous blood PCO_2 plateau after 20-30 seconds showed free mixing throughout the lung bag system. The end-tidal PCO_2 then rises linearly and measurements were made every 20-30 seconds for 4-5 minutes, achieving PCO_2 levels of 70-80 mm Hg. It is fairly assumed that the end-tidal PCO_2 is very close to, or the same as, the central nervous system and respiratory "centres" tissue PCO_2 level. It is considered to be a great advantage that, in this re-breathing procedure, the resultant ventilation no longer affects the alveolar and body PCO_2 levels (open loop). The following results were obtained (means throughout):

	NON-DIVERS	DIVERS	T TEST (P)
VE/PCO_2 (S) 1/min/mm Hg	2.9 ± 0.40 (SD)	1.94 ± 0.20	< 0.05
PCO_2 (VE = 0) (B) mm Hg	39.6	39.4	ns
VE (PCO_2 = 60 mm Hg) 1/min	54.2 ± 5.5	36.3 ± 3.2	< 0.01

In the non-divers the value of 'S' ranged from 0.9 to 7.4 1/min/mm Hg and in the divers from 0.6 to 3.9 1/min/mm Hg. Distribution in both groups was positively skewed.

It was concluded that the carbon dioxide sensitivity of the divers was significantly below that of the non-divers. They also considered that the divers did not represent a distinct population different from the normal one but "rather a group of normal healthy subjects with either an inherent or acquired relatively low CO_2 response." Finally they found that individual carbon dioxide sensitivity did not correlate with either diving experience or current diving activity.

The present author does not propose to discuss the detailed pros and cons of Read's method except to remark that it is a strange procedure to measure the ventilatory response to rising tensions of carbon dioxide while it is being totally frustrated in its purpose and function. The marked inter-individual and intra-individual variation of carbon dioxide sensitivity, as measured by this technique (Rebuck & Slutsky, 1981), suggests that, as in other methods, there are complex and unpredictable events in the nervous system, particularly in such a wholly unnatural physiological situation. Despite claims to the contrary, the ventilatory response during rebreathing is affected by the respiratory rhythm adopted (Rebuck et al, 1974). Rebuck and Slutsky (1981) partly attribute the great variation of carbon dioxide sensitivity in normal subjects to marked individual variation of tidal volume and respiratory frequency. Yet the authors, although continuously monitoring these parameters, make no mention of them, Kerem et al (1980) commented elsewhere on the abnormally slow breathing (skip or controlled) of many divers when connected to a breathing circuit.

The authors claim that these results are similar to those of Florio et al (1979), who used a "high CO_2 first" steady state method. Although the carbon dioxide sensitivity, as measured by slope "S", is of the same order in the two studies, there are conspicuous differences. The non-stimulating PCO_2 (B) in Florio's report is non-divers 32 and divers 35.3 mm Hg. In the present study the figures are 39.6 and 39.4 mm Hg respectively. This difference, is of course, partly due to rebreathing. Further in Florio's study the ventilatory volumes at a PCO_2 of 60 mm Hg (extrapolated) are 77 1/min. in the non-divers and 43 1/min. in the divers. In the present study the respective figures are 54 and 36 1/min. This contrast is a reflection of

the findings being a very considerable function of the method used viz high CO_2 first, steady state v. high CO_2 last, rebreathing, (see Datan et al, 1940).

There is no mention of even slight symptoms of carbon dioxide intoxication during these rebreathing experiments although the end-tidal PCO_2 levels reached were well above 70 mm Hg. One wonders whether the sensations of unreality etc., of early CO_2 narcosis were masked by the music in the subject's earphones.

The authors, who had formidable statistical support, make the important statement that only Froeb (1960) and Florio et al (1979) had sufficiently large groups to warrant statistical conclusions concerning the carbon dioxide sensitivity of divers. The reader will remember that Froeb could find no important difference between the carbon dioxide sensitivity of divers and non-divers and that Florio's conclusions are somewhat jeopardised by the obviously abnormal VE/PET CO_2 plot of his control (non-divers) group.

The present author would again submit that, in this study, the difference in experience of both quiet and stressful breathing from a respiratory circuit (see Rigg et al, 1977) and the almost certain but unspecified difference of respiratory rhythm of the two groups still leaves the issue in some doubt.

In this context it is instructive to consider another group of normal subjects, the Enga people of New Guinea, who also appeared to show diminished sensitivity to carbon dioxide. They were studied by Beral & Read (1971) using Read's rebreathing technique. A portable apparatus allowed the procedure to be carried out in their own environment at 4000 ft. (1300 m) in the Western Highlands. They were non-smokers and most of them were medical assistants at the Baptist Mission Hospital (5 female, 7 male subjects). The control subjects were Caucasians who were studied at sea level in Sydney (6 female, 18 male subjects). Two more Caucasians were investigated in the field (4000 ft.) after a few days "acclimatisation". To eliminate the effect of "psychogenic hyperventilation" the subjects with a ventilation of 15 or more 1/min. in the first half minute were rejected. Numbers rejected in each group were not given.

The Enga subjects showed low levels of CO_2 sensitivity (S) as compared with the Caucasians:

Enga subjects	1.37 ± 0.30 (SD) VE 1/min/mm Hg CO_2
Caucasian subjects	2.51 ± 1.19 (SD) VE 1/min/mm Hg CO_2 ($P < .01$)

Although it is claimed that altitude acclimatisation has a negligible effect on the slope (S) of the hyperoxic CO_2 response line, the ideal experiment would have tested both groups at the same altitude after full adaptation. The Caucasians (24/26) were studied at sea level, the Enga at 4000 ft. The subjects were switched from air breathing to rebreathing. The effect of rebreathing 50% hyperoxic mixture on the Enga subjects, who were naturally adapted to 4000 ft. (18% O_2, PAO_2 75-80 mm Hg) may have decreased the ventilatory response to rising PCO_2 as the body PO_2 also rose. A separate study of the effect of breathing 50% oxygen on the ventilation of each group at their respective altitudes would have been informative and hopefully, reassuring. It is interesting that the two Caucasians subjects studied at 4000 ft. had 'S' values of 2.3 and 1.7.

The cultural and psychological aspects and the inter-reaction between the Caucasian investigators and their Genga subjects would bear some study. Again, holding the nose of the Genga subjects during the rebreathing was a dominating procedure which could tend to "discipline" respiration and reduce ventilation. It is difficult to see why an impersonal nose clip was not used in both studies.

CONCLUSION

The author feels that the evidence presented in these studies does not conclusively support the unlikely thesis that divers become, temporarily or permanently, less sensitive to the ventilatory stimulus of exercise and carbon dioxide. Although there are significant differences between divers and non-divers in a number of studies, this difference may well be due to other factors. The difficulty of finding truly matching control groups of non-divers is considerable, particularly with regard to the divers' far greater experience of respiratory circuits. The less experienced control subjects are certainly more aware of the containment of their respiration and are prone to increased ventilation, as can be seen in several of the above studies. Another problem is the idiosyncratic respiratory behaviour ("controlled" breathing) of many divers, with marked effects on the ventilation, while under observation on a circuit. It is difficult to believe

that divers really breath in this way (see Broussolle et al, 1972, Bradley et al, 1971 above) during their routine diving activities or in everyday life. They would be in an almost permanent state of hypercarbia.

Finally we must return to the consideration of Lanphier's specific proposal that the oxygen-nitrogen mixture diver is far more vulnerable to oxygen poisoning owing to increased gas density, reduced exercise ventilation and increased CO_2 body levels which are tolerated because of reduction of CO_2 sensitivity. He has certainly shown increased end-tidal PCO_2 levels in exercising divers when breathing oxygen-nitrogen mixtures (including air) at 100 fsw and when breathing oxygen or air at 26 and 4 fsw.

Nevertheless there is increasing acceptance that end-tidal PCO_2 measurements do not accurately represent the mean alveolar or arterial blood PCO_2. Lanphier, himself, now takes this view. He stated, with Camporesi, in "Respiration & Exercise", Physiology and Medicine of Diving (1982) "it was widely assumed that end-tidal sampling provided an accurate index of mean alveolar gas composition. This, in turn, was assumed to provide satisfactory indication of arterial blood-gas values. End-tidal PCO_2 values remain useful as non-invasive estimates but they are interpreted with reservation, especially during exertion and at pressure." He also stated in 1987 (personal communication) that he appeared to have had "an unusual concentration" of "CO_2 retainers" in his 1954-1958 investigations. He continued "I would no longer argue that oxygen limits for general use should be conservative enough to be safe for such individuals. I think we must discover and take account of them in other ways." Another important consideration is that, as far as the author knows, there is no reported study of simultaneous arterial blood and end-tidal sampling and PCO_2 measurements in exercising divers at pressure.

Again there have been no convincing signs or symptoms of oxygen poisoning or hypercarbia in any of Lanphier's very large series of oxygen-nitrogen mixture dives to 99 fsw (15 minutes only) described above (Lanphier, 1955, (ii) and 1958). The only instance of undoubted oxygen poisoning, under controlled conditions (Lanphier, 1955, (i)) was the celebrated occasion when an exercising subject convulsed (wet chamber) after breathing (open circuit) 47% ONM at 100 fsw (PIO_2 1.8 ATA, DO_2 29 fsw) for a period of nineteen minutes. This relatively short exposure, which caused such alarm and an immediate dramatic remedy, was, however, within the reported normal range of oxygen tolerance when

breathing pure oxygen at this depth (Donald, 1945).

Finally there is the Royal Navy's and NATO's experience with oxygen-nitrogen mixtures using an oxygen partial pressure limit of 2 ATA in a semi-closed circuit (constant mass flow) without a single case of oxygen poisoning over 50 years.

For all these reasons it is essential to put the matter to the test under controlled conditions and actually demonstrate conclusively, or otherwise, that, apart from end-tidal PCO_2 changes, oxygen-nitrogen mixtures divers suffer from accelerated oxygen toxicity. As already suggested in Chapter 4, a moderate increase of the depth or of the partial pressure of oxygen in the mixture to give equivalent oxygen depths of, say, 1.6 to 2.5 ATA, under safe controlled, experimental conditions, with a significant number of subjects and with pure oxygen "control" dives, would soon show whether there really is an increased danger of oxygen poisoning. As yet neither of Lanphier's hypotheses (possible adverse effect of hyperbaric nitrogen and/or adverse effect of possible carbon dioxide retention on oxygen toxicity) has been proven or convincingly supported. The total lack of any direct demonstration of these purported hazards of oxygen-nitrogen mixture diving, over so long a period, makes it not unlikely that the elaborate and expensive restriction of oxygen-nitrogen mixture diving during the last 35 years in certain countries, has been to avoid dangers which may well not exist. Finally, it is to be emphasized that the putative hazard of *accelerated* oxygen poisoning in oxygen-nitrogen mixture divers is being discussed here and not the established dangers of oxygen poisoning itself.

REFERENCES

BERAL, V and READ, D.J.C. (1971): Insensitivity of respiratory centre in the Enga people of New Guinea. *Lancet*, 2, 1290-94.

BRADLEY, M.E., ANTHONISEN, N.R., VOROSMARTI, J. and LINAWEAVER, P.G. (1971): Respiratory and cardiac responses to exercise in subjects breathing He-O_2 mixtures at pressures from sea level to 19.2 ATA. *Proc. Fourth Symposium on Underwater Physiology, Academic Press, New York and London.* 325-337.

BROUSOLLE, B., BENSIMON, E., and ONJON, J.L. (1969): La sensiblite ventilatoire au gaz carbonique des plongeurs sous-marins. *Compt. rend. des seances Soc. de Biol.*, 163, 1641-2646.

BROUSSOLLE, B., BENSIMON, E., MICHAUD, A. and VAGAZZI, C. (1970): Ventilatory response and alveolar carbon dioxide of divers and non-divers when exercising in hyperbaric atmospheres. *Proc. Third International Conference on Hyperbaric and Underwater Physiology, Marseille: Doin.*, 80-87.

CUNNINGHAM, D.J.C., CORMACK, R.S., O'RIORDAN, J.L.H., JUKES, M.G.M. & LLOYD, B.B. (1957). An arrangement for studying the respiratory effects in man of various factors. *Q. J. Exp. Physiol.*, 42, 294-303

DAHAN, A., BERJENBOSCH, A.A.D., DEGOEDE, J, OLIVIEVIER, I.C.W. and BOVILL, J.G. (1990): On a pseudo-rebreathing technique to asses the ventilatory sensitivity to carbon dioxide in man. *J. Physiol.*, 423, 615-29.

FLORIO, J.T., MORRISON, J.B. and BUTT, W:S. (1979): Breathing pattern and ventilatory response to carbon dioxide in divers. *J. Appl. Physiol.*, 46 (6), 1076-1080.

FROEB, H.F. (1960): Ventilatory response of SCUBA divers to carbon dioxide inhalation. *J. Appl. Physiol.*, 16, 8-10.

GOFF, L.G., BRUBACH, H.F. and SPECHT, H. (1957, (i)): Measurements of respiratory responses and work efficiency of underwater swimmers. *J. Appl. Physiol.*, 10, 197-202.

GOFF, LG. and BARTLETT, R.G. (1957, (ii)): Elevated end-tidal carbon dioxide in trained underwater swimmers. *J. Appl. Physiol.*, 10, 203-206.

JARRETT, A.S.(1966): Alveolar carbon dioxide tension at increased ambient pressure. *J. Appl. Physiol.*, 21, 158-162.

KEREM, D., MELAMED, D. and MORAN, A. (1980): Alveolar carbon dioxide during rest and exercise in divers and non-divers breathing oxygen at 1 ATA. *Undersea Biomed. Research*, 7, 17-26.

LALLY, D.A., ZECHMAN, F.W. and TRACY, R.A. (1974): Ventilatory response to exercise in divers and non-divers. *Respir. Physiol.*, 20, 117-129.

PAPPENHEIMER, J.R. (1944): Respiratory requirements during exercise. Handbook of Respiratory Data, Nat. Res. C, Comm. *Aviation Med. Res.*, Washington.

READ, D.J.C. (1967): A clinical method for assessing the ventilatory response to carbon dioxide. *Australian Ann. Med.*, 16, 20-23

REBUCK, A.S. and SLUTSKY, A.S. (1981): Measurements of ventilatory response to hypercapnia and hypoxia. Regulation of Breathing Ed. T.F. Hornbein, Dekker, *N.Y. & Basel, Part II.*, 745-772

REBUCK, A.S., RIGG, J.R.A., KANGALEE, M and PENGELLY, L.D. (1974): Control of tidal volume during rebreathing. *J. Appl. Physiol.*, 37, 475-8

RIGG, J.R.A., INMAN, E.M., SAUNDERS, N.A., LEEDER, S.R. and JONES, N.L. (1977): Interaction of mental factors with hypercapnic ventilatory drive in man. *Clin. Sci. Mol. Med.*, 52, 269-75

SALZANO, J.K., RAUSCH, D.C. and SALTZMAN, H.A. (1970): Cardiorespiratory response to exercise at simulated sea water depth of 1000 feet. *J. Appl. Physiol.*, 28, 34-41.

SALZANO, J.V. BELL, W.H., WEGLICKI, W.B. and SALTZMAN, H.A. (1966): Metabolic and haemodynamic responses to exercise at increased pressure. *Proc. Third Symposium Underwater Physiology, Nat. Res.Council, Washington.* 351-360.

SCHAEFER, K.E. (1955): Role of carbon dioxide in the physiology of human diving. *Proc. Underwater Physiol. Symposium. Pubn. 377, Nat. Ac. Sc. and Nat. Res. Council.*, 131-138.

SCHAEFER, K.E. (1965): Adaptation to breath-holding. *Pubn. 1314. Nat. Ac. Sc. and Nat. Res. Council, Washington.* 237-251.

SHERMAN, D., EILANDER, E., SHEFER, A and KEREM, D. (1980): Ventilatory and occlusion and responses to hypercapnia in divers and non-divers. *Undersea Biomed. Res.*, 7, 61-73.

SONG, S.H., KANG, D.H., KANG, B.S. and HONG, S.K. (1963): Lung volumes and ventilatory responses to high CO_2 and low O_2 in the ama. *J. Appl. Physiol.*, 18, 466-70.

APPENDIX 1

THE ADMIRALTY EXPERIMENTAL DIVING UNIT

After serving as Flotilla Medical Officer in the Second (Captain Warburton-Lee VC., RN.) and Fifth (Captain Lord Louis Mountbatten, D.S.O., RN.) Destroyer Flotillas I was posted to HMS *Forth* in Holy Loch. The ship serviced submarines patrolling in the North Atlantic, the Arctic and the North Sea. It was a fascinating experience to meet and work with these extraordinary men. However, although Holy Loch was a beautiful and serene place there was, apart from routine duties, little to do except read, meditate and, occasionally, walk about.

Thus, in March 1942, after volunteering for 'special service', whatever that meant, I found myself proceeding to HMS *Dolphin* (Fort Blockhouse, Gosport). I soon discovered that I was to be associated with underwater activities which were quite apart from those of the submarines based there. The reasons for these developments were highly confidential. The Italian Navy, which has always had a penchant for unusual and gallant underwater operations, had, in December 1941, struck a critical blow against the Royal Navy by severely damaging the two battleships HMS *Queen Elizabeth* and HMS *Valiant* in Alexandria harbour. Both ships were rendered non-operational for a considerable time. The harbour was shallow and lists were rapidly controlled. They successfully deceived the enemy by behaving as if nothing had happened. This was possible as all the human torpedo crews involved had been captured. All drills, shore leave, receptions and band parades continued with due pomp and ceremony. 'Steam' was kept up as if ready to sail at short notice.

This brilliant attack was made by specially designed torpedos which were ridden and controlled by two divers. The powerful warhead, with neutral buoyancy, could be detached from the torpedo and clamped onto the ship's bottom or laid on the seabed. The torpedoes were carried to

within a few miles of the harbour by a parent submarine. This was by no means the first of such underwater operations. A similar planned assault on Alexandria harbour in August 1940 had been unwittingly frustrated by the bombing and sinking of the Italian parent submarine *Iride*. In November, 1940, three 'human torpedos' had launched an attack on HMS *Barham* in Gibraltar harbour. Only one of three craft penetrated the harbour defences. It was ridden solo by Lieutenant Brindelli who had ordered his number two to 'bale out' after respiratory difficulties. Brindelli came within 100 yards of the *Barham* when his torpedo engine failed. He pulled the inert torpedo to within 30 yards of the ship when he suffered breathing apparatus failure and probably, carbon dioxide intoxication. This remarkable man survived and was only captured as he nearly succeeded in boarding a Spanish ship. A similar attack on Gibraltar harbour in September 1941 resulted in the destruction of two large tankers and a cargo vessel. The Italian Navy also had underwater swimming teams who were attaching limpet mines to ships in different harbours with increasing effect.

These events had not gone unnoticed by 'A Former Naval Person' and a brisk signal went to the Chiefs of Staff in January, 1942: "Please report what is being done to emulate the exploits of the Italians in Alexandria harbour . . . Is there any reason why we should be incapable of the same kind of scientific aggressive action that the Italians have shown?"

At long last Their Lordships moved and ordered the development of 'human torpedos' and other unorthodox methods of underwater attack. It was estimated that we were at least seven years behind the Italians in this field, although new types of midget submarines were already being developed. Flag Officer, Submarines, Admiral Max Horton, D.S.O. was placed in charge of all these activities with the support of HMS *Excellent* (Gunnery and Diving School, Portsmouth). Flag Officer, Submarines was represented in this regard at HMS *Dolphin* by Commander G.M. Sladen, D.S.O., RN.

On my arrival at Blockhouse I was put through refresher courses of air diving and submarine escape followed by oxygen diving in Horsea Lake. It was here that the first British 'charioteers' were being trained. They were using a self-contained oxygen breathing apparatus which had a well-designed rubber counterlung and a large radial carbon dioxide absorbent canister (4.5 lbs). Although it had been developed with

considerable urgency it appeared to be more robust and safer than the captured Italian models. Oxygen breathing was essential in these covert underwater attacks on ships and harbours so that no gas needed to be vented and the risk of detection minimised.

Author (centre) with two members of the first charioteer group after a day's oxygen diving in Horsea Lake, Portsmouth, April 1942.

My duties as a humble Surgeon Licutenant were simple. I attended all oxygen diving carried out by future charioteers in case anyone 'flaked' or suffered other misfortunes. With regard to the safety times and depths when breathing oxygen under water, it was tacitly assumed that these could be directly inferred from the oxygen tolerance determined in the 'dry' hyperbaric chambers when the resting subject breathed oxygen in compressed air. The safety limits at this time (1942, Royal Navy and US Navy) were of the order of 2 hours at 50 feet and 30 minutes at 90 feet of sea water (see Chapter 1). In April and May there were a number of incidents at Horsea Lake (maximal depth just over 25 fsw) when oxygen divers 'did not feel right' or were transiently dissociated. It was felt that minor symptoms were inevitably in groups of men who had not dived before and were under intense training. Again, the mouthpiece, noseclip and soft helmet 'set-up' of the "human torpedo" oxygen diver was far more

claustrophobic than air diving with a large, rigid and windowed helmet in which fresh air sweeps constantly past the face. Then, suddenly, an oxygen diver was lost at these relatively shallow depths. The precise events leading to his death were not fully determined but there was a strong suspicion that oxygen poisoning had occurred. There was a growing realisation that it was desirable, if not essential, to have a constant background of careful investigation to ensure the maximal safety of these men during training and during their dangerous covert operations.

In May, 1942 the Royal Navy took over the experimental wing of Siebe, Gorman and Company at Surbiton. This exceedingly well equipped unit contained a large open 12 foot tank, a smaller open 25 foot tank, a 'wet' pressure chamber (the 'wet pot') and several dry pressure chambers of different sizes. Expert technical help was immediately available. Every member of the company from Sir Robert Davis and his sons to the nurse in the sick bay provided all the assistance possible. It had been decided to test all operational personnel (charioteers, X-craft (miniature submarines) crews, underwater swimmers etc.) in controlled and reasonably safe conditions while breathing oxygen underwater at increased pressures in the wet pressure chamber. I travelled daily from Portsmouth to Surbiton to act as medical officer in these trials and saw experimental diving in hyperbaric chambers for the first time. It was a strange scene as divers were lowered into the 'wet pot', particularly as a considerable number were hauled out unconscious after convulsing. On occasions, the next diver stepped over the last casualty to take his turn. There was an air of extreme urgency as the planning and method of attack on enemy harbours and the proper defence of our own, depended on a full knowledge of how deep these oxygen divers, friendly or hostile, could go with reasonable safety. The grand mal types of convulsion occurring made it absolutely clear that we were encountering acute and severe oxygen poisoning at depths and in times then considered quite safe by all authorities.

After about a fortnight of this unusual and occasionally chilling experience I received a 'chit' from Their Lordships instructing me to proceed forthwith to some obscure posting in the Shetland Islands. Next day I was travelling for the last time from Portsmouth to Surbiton by train and, by sheer chance, Commander Sladen came into the same compartment. Sladen, who had played rugby football for England on a number of occasions, was a man of unlimited energy and quick decisions. He was a

highly successful and much decorated submarine commander having been in many actions, including the torpedoing of the heavy German cruiser, the *Prinz Eugen*. I informed him of my new appointment and must have shown my regret at leaving this brave company. He asked me point-blank if I would like to be responsible full time, for the safety of the subjects and for the investigations and programme at the Unit. I replied that I certainly would. "Give me your chit" he said, "You'll hear no more from Their Lordships." Nor did I.

Thus, there I was, a Surgeon Lieutenant, aged 30, with no research and little hyperbaric experience, with a not inconsiderable research unit on my hands. I was, by some strange and splendid accident, in no way responsible to any senior medical or scientific person or committee and apart from operational demands, I was given a carte blanche in programme and supplies. My immediate responsibility was to Commander Sladen and Commander W.O. Shelford, RN., Submarine Escape Officer and later Superintendent of Diving. In a relatively short time the staff of the Unit expanded and consisted of Commissioned Gunner Mr. E. Crouch, RN., two diving Chief Petty Officers, a gas analyst, a typist secretary and a variable number of hands for diving, routine and maintenance.

A further piece of extreme good fortune was the discovery that J.B.S. Haldane (fils) was working in one of the dry chambers for two days a week completing a contract to investigate the physiological factors relevant to the submarine HMS *Thetis* disaster in 1939, (Alexander et al, 1939; Case & Haldane, 1941; Haldane, 1941). During the next year, while Haldane was completing his programme, he gave most valuable advice particularly during the development of oxygen-nitrogen mixture diving (see Chapter 5) and also on the statistical treatment of the Unit's early studies of oxygen poisoning (see Chapter 2).

In August, 1942, in addition to the many operational personnel passing through the Unit as subjects, we were joined by a team of twelve carefully selected volunteers. These men also acted as experimental subjects, particularly in longer term projects. The Unit programme was rapidly becoming engaged in more and more operational problems. We needed a larger staff who could service and maintain respiratory and other apparatus, who could dress the divers and who could help to attend and observe subjects both in the open water and in chambers. Care was taken to ensure that they were briefed and involved in all Unit activities. One of

my most pleasant memories was of an Able Seaman showing a senior visiting academic how to handle a wayward spirometer during marked pressure changes. The teacher and pupil were completely engaged without a trace of disrespect or false dignity. Another visitor wrote some years later "In spite of the risk and unpleasantness of the job, the experimental department was always a scene of cheerful activity." Some of these divers transferred to operational teams and some operational personnel came back into the 'experimental' group. This leavening both ways was good for morale and increased efficient communication with those in the field.

While ordering notepaper for the Unit I decided that we fully deserved a more definite and prestigious identity. I adopted an Admiralty crest and titled ourselves the Admiralty Experimental Diving Unit. During the next three years we were in constant touch with the Admiralty over a host of problems. They also sent many VIPs, including those of our allies, to see our work. No-one objected to our splendid self-bequeathed title.

In the large study of oxygen poisoning in divers, which is fully described in Chapter 2, it was necessary for the observers to assess the various symptoms and signs reported by the divers. Convulsions are the only unequivocal end-point of oxygen toxicity but other symptoms, even lip twitching in a soft helmet, are largely a matter of report. The morale and reliability of these men were therefore critically important. They were well aware that the success of future operations and the safety and competence of operational personnel completely depended on the accurate and faithful reporting of symptoms in these urgent investigations. When we first transmitted our findings elsewhere concerning the quite unexpected and marked increase of oxygen toxicity under water, there was, initially, almost total disbelief. Most unfortunately, mention must have been made by some unknown persons of possible "feigned symptoms". Such remarks were not only unforgivable but die hard. Indeed, the author only learned of this recently when, to his astonishment, he was asked, in writing, by a US research worker 45 years later "which was the study spoiled by feigned symptoms?" It is therefore necessary to point out, somewhat reluctantly, that the subjects in this oxygen study, including members of the experimental team, received many gallantry awards (Victoria Cross, 4; George Cross, 5; Distinguished Service Order, 10; Distinguished Service Cross, 21; Distinguished Service Medal, 8; George Medal, 5; Conspicuous

Gallantry Medal, 5; British Empire Medal, 16, etc). It is most unusual, if not unique, for an investigator to be able to produce such tangible evidence of the intrepid mould and resolution of his subjects. The extraordinary fantasy of such brave and responsible men feigning convulsive attacks and other signs and symptoms in front of their colleagues and medical officer in these critical trials does not merit polite consideration.

Oxygen-nitrogen Mixtures

In October 1942, it was decided that, as oxygen diving was so dangerous below 30 fsw, it was necessary to develop the use of oxygen-nitrogen mixtures in self-contained counterlung breathing apparatus for use at greater depths. The oxygen breathed could be diluted with nitrogen, although not so much as in air, and this would allow diving to considerably greater depths without the risk of oxygen poisoning.

The midget submarines were now working up to operational efficiency. In their harbour-attack procedure, a member of the crew might be required to lock out of the craft, surface and inspect or even cut defensive nets and then return to the submarine and lock back in. This might be necessary at depths greater than 30 fsw and such oxygen-nitrogen mixtures would be very useful in this context as oxygen poisoning could be avoided. The development of oxygen-nitrogen mixture counterlung breathing apparatus is discussed in some detail in Chapter 4. This work was completed and reported in March 1943 (Donald 1943 (i)).

In May 1943, counter-mining officers, on entering Bizerta, Tunisia, found many large German mines with six-day clocks for use in harbours and basins. These had no magnetic or acoustic units and could not be detected and 'swept' by the usual methods. This meant that it would be difficult, if not impossible, to guarantee the safe handling of troop and supply ships in newly captured ports. Unless some means of locating these mines were found, the planned invasion of Europe was in jeopardy. After considerable consultation and much rumination, it was proposed by Officer-in-Charge of Counter Mining that the only way to deal with this problem, was, if possible, to organise large teams of divers to search for these mines systematically. Commander John Stuart Mould, G.C., G.M., RANVR. (called 'Mouldy', of course) came to see me about 'the problem'. Mouldy had achieved considerable distinction in many dangerous counter-mining

operations which, to quote the *London Gazette*, "included the recovery, rendering safe and investigation of the first German magnetic/acoustic unit and moored magnetic mines." Oxygen diving was out of the question as depths up to 80 fsw would be encountered. Thus, a few weeks after completing the first mixture counterlung development, an unexpected and critically important use for this apparatus and technique presented itself.

A number of large teams of divers were required and it was essential that they were highly mobile and able to dive and search anywhere at short notice. Each diver moved on bottom along a jackstay on a grid, searching one side and then, on return, the other. It was necessary to test the efficacy of oxygen-nitrogen mixtures even further and to assess the feasibility of large teams using this not altogether simple procedure requiring scrupulous supervision. During 1943 P Party personnel (P for Port Clearance) were trained at the Admiralty Experimental Diving Unit, particularly in the care and maintenance of mixture breathing apparatus. They also acted as attendants and subjects in many experimental dives 'in the dry' and 'in the wet'. Officers were taught the theory and calculations involved in mixture diving. It was felt strongly that this considerable degree of understanding and involvement was essential for the reliable use of mixtures by counterlung divers in demanding and dangerous operational conditions. It was a great pleasure to work with these brave and intelligent men, whose sense of humour was never far below the surface. In thousands of oxygen-nitrogen mixture dives in Europe after D-day by P Parties, there was no instance of oxygen poisoning, oxygen lack or decompression sickness. Their quite remarkable operational record in Europe is well documented (Grosvenor and Bates, 1956). Their first task was to clear Cherbourg harbour for the US forces. General Eisenhower sent a warm signal of thanks. They worked their way along the coast of Northern Europe, sometimes by-passing unfinished battles and uncaptured ports to return later. The clearing of Antwerp harbour and its approaches was a tremendous undertaking, much of it carried out under enemy fire. Over eight and half million square feet were searched. Rotterdam, Hamburg and Bremen were finally cleared. Bremen was to be the port for the US occupation zone and its clearance was the greatest task of all. All six British P Parties and the Dutch P Party (trained in the United Kingdom) took part in this operation. It is estimated that 9,500,000 square

feet of docks and harbours in Bremen were searched and cleared. These were certainly days of high courage and great achievement.

Original Port Clearance counterlung mixture diver under way.

Underwater Oxygen Swimmers

In 1930, Commander de Carlieu designed and introduced rubber swim 'fins' attached to the feet. These were widely adopted by sportsmen, some of whom carried air cylinders giving a steady flow of air into their mask. The adoption of a closed circuit with oxygen breathing and without venting was ideal for covert underwater fin-swimmers and was used by all combatants. These divers were, of course, known to the public as frogmen. A 'crawl' leg stroke provided the propulsion, the two swim-fins and legs imitating the driving action of a dolphin's tail.

In view of our recent findings, these oxygen swimmers did not go deeper than 25 feet, except for very brief periods and only for urgent operational reasons. The use of oxygen-nitrogen mixtures was carefully considered but rejected. The higher flow required would have necessitated considerable venting of gas with greater risk of detection. Endurance would have been

reduced and the large cylinders needed would have impaired mobility and stream lining. Water turbulence was most undesirable as it, too could alert harbour and coastal defences.

The Royal Navy frogmen often worked in northern waters and needed protection from the cold. Their beautifully designed and streamlined rubber suits were the most efficient and elegant ever produced. The main contributors were W. Gorham of the Dunlop Rubber Company and Commander Shelford. Colonel H. Hasler, D.S.O, R.M. (of 'cockleshell' fame) and Lt.Cdr. B. Wright, RCNVR. an experienced spear fisherman, both stimulated and helped their development. These underwater swimmers were needed for a host of operational requirements, the sabotage of ships, docks and bridges (limpeteers), boom and ship defence units, beach reconnaissance (the frogman could slip off his mask and carry his flippers), and so on.

In the early days of training these frogmen encountered a new and terrible danger called shallow water blackout (SWBO). These blackouts occurred at depths of 20 fsw or less. Several swimmers were lost. Those who surfaced safely described how they had become dissociated and even unconscious. The investigation and elimination of SWBO is described in detail, with references, in Chapter 5. Briefly it was discovered that these swimmers could have oxygen uptakes and carbon dioxide production far beyond those ever encountered in booted divers. This led to CO_2 absorbent canister overload and the divers being anaesthetised by their own carbon dioxide.

At this time increasing interest was being taken in the oxygen uptake of divers as the safety of both oxygen divers and oxygen-nitrogen mixture divers depended on this being accurately known. In the early development of mixture diving, a somewhat approximate estimation of oxygen uptake had been obtained by balancing the oxygen supply with the oxygen uptake in a steady state at rest and during various degrees of exertion. A new and far more accurate method was developed. This depended on a demand valve* fed through a reducing valve and a long intermediate pressure line to the diver's counterlung. No venting was allowed and the diver's face,

* The demand valve, which releases air or other gases to the diver's breathing apparatus when a slight negative pressure is generated on inspiration, was, astonishingly, invented and used in an independant diver's breathing apparatus by Rouquayroll and Denayruse as early as 1865

mouthpiece and nose clip could usually be observed. The oxygen was supplied from a relatively small cylinder in which exact changes of pressure could be read by the observers at the surface, using a very sensitive and accurate pressure gauge (Donald and Davidson, 1944). This new method could be used in any conditions even in open tidal water and was ideal for determining oxygen uptake (P Party divers, frogmen, etc.), under operational conditions. The very high oxygen uptake of frogmen swimming at speed, already mentioned, was a great surprise to us. Looking back, of course, it should have been obvious that the liberation of the diver's legs from his heavy restraining boots to free swimming would inevitably bring many more powerful muscles into play.

In late 1943 and early 1944 the Unit carried out a large programme of dives to determine oxygen tolerance at relatively shallow depths (25 to 40 fsw). The effect of exercise and temperature on oxygen tolerance underwater was also carefully studied at various depths. The great variability of tolerance in individuals and between individuals, already demonstrated, made large series essential.

A system for diving on oxygen to greater depths in covert operations was evolved at this time. Relatively short stays at 'toxic' depths (50 to 70 fsw) were alternated with brief intermissions near the surface (Donald cit. Admiralty Fleet Order 4565,1944). This potentially valuable technique has not been used operationally since that time.

As D-Day approached we felt ready for all predictable requirements for a major assault from the sea. There were some last-minute requests, such as a light escape apparatus for the crews of amphibious tanks and other heavy vehicles that might be sunk on the way in. The part played by the underwater section of the Royal Navy and Royal Marines in the invasion of Europe is little appreciated. Beach reconnaissance frogmen who determined the slope, the depth of approach and suitability for tanks and heavy vehicles, including sampling, were landed surreptitiously before D-Day from miniature and ordinary submarines and from light surface craft. The formidable array of obstacles to prevent landings, both on the beach and underwater, were located and studied in detail. These obstructions could, of necessity, only be demolished just before landing, usually under heavy enemy fire. The ten gallant teams (6 RN, 4 RM) of Landing Craft Obstruction Clearance units performed one of the most critical and dangerous tasks of the whole war. Miniature

submarines were submerged about three miles off the coast and surfaced at the agreed time to flash hooded lights seaward to guide the assault. The Port Clearance Parties were close behind to search and clear all docks, basins, harbours and canals and to check lock gates and bridges for mines. The harbours, both old and new, were, in turn, protected by the Royal Marine Boom Defence Units who also patrolled under water.

At home the transfer of P Party Headquarters to Brixham gave the Admiralty Experimental Diving Unit better facilities for even more realistic operational trials in the harbour and open sea. In August 1944, Surgeon Lieutenant Commander W. Davidson, RN. joined the Unit to train in hyperbaric work and gave valuable help. As victory in Europe became more certain we turned our attention to a number of new problems. Lighter swim suits were developed and there was a sudden interest in the dangers of the Pacific and other Far Eastern seas. We were able to commence a number of projects that had been set aside because of unremitting operational demands.

One of these projects was to determine the margins of safety in the method of Surface Decompression being used somewhat tentatively by the Royal Navy in air diving during World War II. In this procedure the diver came straight up from the bottom to the surface at about 100 feet per minute. His helmet and weights were removed as quickly as possible (1.5 minutes maximum). He was then recompressed in a deck chamber to his original depth. The maximal total time allowed from 'bottom to bottom' was five minutes. After another five minutes at maximal pressure a standard decompression (air and oxygen) was carried out.

It has since been claimed (Davis, 1951) that this technique had been used during diving on the *Empress of Ireland* (190 fsw) in 1914 and on the *Laurentic* (130 fsw) from 1917 to 1922. In fact the most careful enquiries reveal that immediate recompression and subsequent decompression had only been used after emergency surfacing due to 'blow up' or other severe accidents or illness, as was the case in air diving the world over. The first deliberate, planned use of *immediate* surface decompression in air diving originated in HMS *Tedworth*, the Royal Navy deep diving vessel, during World War II. A table was developed with maximal time allowed on bottom ranging from 50 minutes at 120 fsw to 15 minutes at 250 fsw. The surface decompression procedure and table were not even mentioned in the Royal Navy diving manual of that time, nor had its overall safety ever

been tested and approved. The attendant dangers of almost immediate fatal decompression sickness or of irreversible paralyses were indeed fearsome. Consultation with diving officers of that period revealed that the procedure had only been used on a few important occasions at the "lesser" depths (120 to 200 fsw). Not surprisingly, urgent requests were now being made (1945) that the procedure and table should be formally examined and tested before further use.

In these 1945 Admiralty Experimental Diving Unit trials (Donald & Davidson, 1945), for obvious reasons, the first surface decompression profiles were performed in compressed air using goats as subjects. Human chamber dives were then carried out, followed by full sea dives from HMS *Tedworth*. These investigations showed that it was highly dangerous to use this method with exposures greater than 20 minutes at 190 fsw.

The summary of recommendations made after this investigation were as follows (Donald & Davidson, 1945):

1. The 'Tedworth Method' of Surface Decompression is safe for the following depths and times on the bottom:

Depth	Time on Bottom
Up to 130 fsw	50 minutes
130 - 150 fsw	40 minutes
150 - 170 fsw	30 minutes
170 - 190 fsw	20 minutes

2. These times should never be exceeded. If this occurs inadvertently. e.g. the diver is fouled, then the stops must be carried out on the shot rope.

3. This method should be employed only by *TRAINED* personnel in a properly equipped diving ship, as speed in recompressing the diver is essential.

4. Oxygen breathing is essential during decompression from the 60 fsw stop to surface.

5. This method should not be employed at depths greater than 190 fsw owing to the grave risk of *"CHOKES'* on rapid surfacing."

Air diving is now restricted to 50 msw (165 fsw) but surface decompression is still practised a great deal, especially in 'shallow' oil and gas fields. Recompression to the full depth is not necessary. The main dangers of this procedure are always present if there is any slackening of the rigid discipline of accurately measured and restricted 'times on bottom' and there is the slightest leisureliness and resultant lengthening in 'bottom to bottom' time. Rapid handling of the diver on deck requires ballet-like skill and precision. Any clumsiness or carelessness causing delay at surface is particularly hazardous.

We next turned our attention to the ever-present problem of submarine escape. The number of successful escapes from sunken submarines during World War II, even from moderate depths, had been disappointing. The hazards of main compartment flooding, followed by serial escape through a trunk or escape lock were now realised to be great at shallow depths and appalling at greater depths. The breathing of compressed contaminated air, followed by the breathing of oxygen near equalisation and during escape, was full of dangers which were compounded in a stressed and exhausted group of survivors.

Experience before and during the war has shown that the majority of survivors, particularly from greater depths, were not wearing any breathing apparatus and had escaped from air locks or compartments that had equalised with the outside pressure during or after the incident. A whole new look at submarine escape was patently necessary. We proposed to imitate and exploit these successful but somewhat fortuitous escapes without breathing apparatus from air locks (Donald, Davidson and Shelford AEDU Report XVIII, May 1946, Published J. Hyg. Camb. 1948). The air breathing escape procedure to be adopted was as follows:

Stage 1: Escaper enters the relatively small escape compartment and pressure is raised by flooding to the pressure at which the submarine is lying. An air lock is maintained. Time: 2 minutes approximately.

Stage 2: At full pressure, manipulating and opening hatch and emerging from submarine. Time: variable.

Stage 3: Free ascent from submarine to surface at approximately 2 feet per second. Time: varies with depth, 2-2.5 minutes. The rate of ascent had been determined in open water trials by Commander Shelford.

In view of the considerable risk of irreversible decompression sickness, goats were used in these initial experiments which were conducted in compressed air. Escape profiles were carried out from 150 to 300 fsw. Time at full pressure (Stage 2) was varied from 3 to 7 minutes. Symptomless escapes with no decompression sickness were successfully concluded with up to 5 minutes at maximal pressure (Stage 2) at 150, 200 and 250 fsw. The use of oxygen-nitrogen mixture instead of air (33% oxygen; 67% nitrogen) allowed safe escape profiles from 300 fsw with up to 7 minutes at full pressure. In these experiments the time at full pressure before escape was deliberately excessive. As regards decompression sickness, it was felt that escapes by this method, with a short Stage 2, were feasible from far greater depths. The problem as to whether the escaper could continue to vent and not inhale while ascending from these great depths was yet to be investigated.

Another important question was whether nitrogen intoxication would jeopardise the competence of the escapers. A series of cancellation tests were performed during and after very rapid compression to 300 fsw. It was shown that compression to 300 fsw, in times as short as one minute, did not cause undue psychological disturbance although there was a slight euphoria in several instances. The cancellation tests showed that the subjects were able to concentrate moderately well during compression and after arrival at full pressure. A few subjects showed marked slowing but maintained accuracy and others lost accuracy without slowing. No subject stopped his test or lost control.

All these air breathing submarine escape experiments were performed in 1945 between VE day (8th May) and VJ day (14th August), a good augury, we hoped. The Ruck-Keene Committee on submarine escape (August 1946) supported this new method of escape and recommended further research and development with a view to its adoption, if feasible. A whole series of animal and human experiments in the late forties, fifties and sixties culminated in escapes in the open sea from a submarine lying on the sea bottom at 600 fsw (182 msw) in 1970. To gauge the progress made, in

the 600 fsw escapes the compression time was 33 seconds, the full pressure period 3 seconds and the time of ascent, when most nitrogen is absorbed, (rate 8.5 feet per second) 68 seconds. The great increase in the rate of ascent was largely and somewhat fortuitously due to the development of a survival suit with a hood and a buoyancy stole to prolong survival at surface. Thus the escaping crew were compressed from atmospheric pressure in the submarine to 600 fsw (19 ATA), locked out and ascended to the surface and atmospheric pressure in the astonishing time of 104 seconds. The twenty-five years work to make this feat possible has been described and reviewed by the author elsewhere (Donald, 1970, 1979 and 1991). These two reports made it clear that, although successful escapes were now possible from 600 fsw and even deeper, there were considerable attendant risks which were only justifiable in real escapes from disabled submarines. Calculations had shown an undesirable degree of nitrogen super-saturation of various 'fast' tissues on surfacing after deep escapes (500 to 700 fsw). It was considered highly probable that there were free bubbles of nitrogen in the circulation after such escapes. For these reasons it was recommended that further deep escape trials should be approached with considerable caution and that repeated deep escapes at relatively short intervals by a single individual should be avoided.

The submarine escape study was my last project. I left the Royal Navy in November, 1945. The Admiralty Experimental Diving Unit at Siebe, Gorman, Surbiton was shut down shortly after the war and the remaining staff returned to HMS *Vernon*, Portsmouth, where the diving headquarters and school were now established. It had been an interesting and concentrated three and a half years.

REFERENCES

ALEXANDER, W., DUFF, P., HALDANE, J.B.S., IVES, G. and RENTON, D. (1939): After effects of exposures of men to carbon dioxide. *Lancet*, 2, 419-420.

CASE, E.M. and HALDANE, J.B.S. (1941 (i)): Human physiology under high pressure. *J. Hyg. Camb.*, 41, 225-249.

DAVIS, R. (1951): Surface Decompression. *Deep diving and Submarine Operations. Fifth Ed. St. Catherine Press, London*, 97-99.

DONALD, K.W. (1943 (I)): Low supply of mixtures in counterlung type of breathing apparatus. *Admiralty Experimental Diving Unit (R.N) Report VII/43.*

DONALD, K.W. (1970): A review of submarine escape trials from 1945 to 1970 with particular emphasis on decompression sickness. *Royal Naval Personnel Committee U.P.S.* 290. Recently published DONALD, K.W. (1991) *J. Roy. nav. med. Serv.* 77, 171-200.

DONALD, K.W. (1979): Submarine escape breathing air. A review and analysis of experiments by the Royal Navy. *Bull. europ. Physiopath.resp.* 15, 739-754.

DONALD, K.W. and DAVIDSON, W.M. (1944): Oxygen uptake of divers. *Admiralty Experimental Diving Unit (R.N) Report XV/44.* Later published: DONALD & DAVIDSON (1954). Oxygen uptake of booted and fin swimming divers. *J. Appl. Physiol.* 7, 31-37.

DONALD, K.W. and DAVIDSON, W.M. (1945): Surface Decompression. *Admiralty Experimental Diving Unit (R.N) Report XVII/45.*

DONALD, K.W., DAVIDSON, W.M. and SHELFORD, W.O. (1946): Immediate surfacing from increased pressures with a view to submarine escape. *Admiralty Experimental Diving Unit (R.N) Report XVIII.* Later published (1948) DONALD, DAVIDSON & SHELFORD, Submarine escape breathing air. *J. Hyg. Camb.*, 46, 176-183.

GROSVENOR, J. and BATES, L.M. (1951): Open the Ports. The story of the human minesweepers. *Wm. Kimber, London.*

HALDANE, J.B.S. (1941): Human life and death at high pressure. *Nature*, 148, 458-460.

APPENDIX 2

TABLE 1

OXYGEN POISONING AT 90 FEET IN THE DRY IN 37 SUBJECTS

Name	Early symptoms	Time of Onset of Early Symptoms (min)	Symptoms at Final End Point	Time of End Point (min)	Age (yrs)	ht. (ins)	wt. (lbs)
Derrick	Slight vertigo	43	prolonged dazzle, severe spasmodic vomiting	96	37	72	120
Sims	Slight twitch left upper lip	55	Severe lip twitching	67	23	70	190
Harman	Dilatation of pupils (sign)	23	Euphoria & lip twitching	62	23	65	141
Goldsworthy	Tremor left upper arm	38	Nausea and vertigo	62	34	64	118
Whittington	Fine tremor of lips	37	Severe lip twitching	54	18	64	143
Brown	Slight lip twitching	7	Dazzle and lip twitching	51	21	66	140
Warren	Drowsiness & pouting of upper lip	1	Blubbering of lips and fell asleep	50	30	69	146
McInnes	Cheek tremor	4	Dazed & lip twitching	50	36	63	122
Fraser	Slight lip twitching	7	Nausea, vertigo, lip twitching	34	19	66	131
Ward	Slight facial twitch	25	Convulsed	33	23	69	148

Name	Early symptoms	Time of Onset of Early Symptoms (min)	Symptoms at Final End Point	Time of End Point (min)	Age (yrs)	ht. (ins)	wt. (lbs)
Murton	Fine twitch of lips	18	Convulsed	32	20	72	166
Dickie	Fine twitch upper lip	3	Severe lip twitching	32	21	68	150
Shields	No warning	–	Convulsed	30	20	67	142
Wadsley	Slight twitch of lips	11	Convulsed	26	35	66	164
McAtamney	Drowsiness	1	Drowsiness lip twitching	25	25	69	147
Robertson	Slight tremor left upper lip	18	Severe lip twitching	24	25	65	161
Wallis	Sensation of epigastric tension	1	Lip twitching	23	23	67	142
Gray	Fine twitch right upper lip	10	Lip twitch, twitch left arm Amnesia	20	34	70	138
Martin AB	Slight lip twitching	–	Convulsed	19	24	69	138
Rogers	Slight twitch upper lip	10	Vertigo & Severe lip twitching	18	25	67	169
Martin	Sensation of epigastric tension	6	Vertigo plus plus	18	21	67	143
Smith	No warning	–	Lip twitching	17	23	67	154
McCourt	–	–	Lip twitching Spasmodic respiration	17	23	68	159

Turner	Fine twitching of lips and pallor	10	Lip twitching Spasmodic respiration	17	40	70	157
McLaughlin	Lip twitching	1 3/4	Lip twitching	16 1/4	19	69	111
Mould	Slight lip twitching	10	Severe lip twitching & spasmodic respiration	16	33	71	154
Cullen	Inspiratory 'predominance'	8	Lip twitching and syncope	15	32	67	145
Donald	Nausea and weakness	5	Syncope and confusion	15	31	73	165
Green	Pins and needles in hands	12	Lip twitching	14	19	69	149
Knight	Twitch left lower lip	1	Lip twitching	12	18	64	125
Tennan	Stinging sensation beneath eyes	2	Dazed & lip twitching	9	27	72	146
Mulberry	Fine twitch lips		Lip twitching & vertigo	9	23	69	143
Gray	Twitch upper lip	2	Severe lip twitching	7	21	67	141
Gibson	Slight twitch upper lip	2	Diaphragmatic spasm	7	23	68	133
Kirk	Fine twitch both lips		Severe nausea	6	21	68	133
Williams	Slight tremor of lips	2	Severe lip twitching	6	23	71	166

TABLE 2

DIVES TO 50 FT. IN THE WET. LIMIT OF TIME 30 MINS.

No.	Time	Symptoms	Work done
1	30	No symptoms	Exercised with 2-7 lb weights
2	25	CONVULSED	10 mins. with weights
3	30	No symptoms	Mild exercise
4	14	CONVULSED	None
5	21	Lip twitching	Mild exercise
6	12	Malaise	5 mins. gently with weights
7	30	No symptoms	Mild exercise
8	9	Lip twitching	Mild exercise
9	10	Lip twitching	Mild exercise
10	30	No symptoms	Hard exercise
11	11	Nausea and respiratory distress	Mild exercise
12	13	CONVULSED	None
13	18	CONVULSED	Mild exercise
14	16	Lip twitching	Intermittent exercise
15	28	Lip twitching	Mild exercise
16	30	No symptoms	Mild exercise
17	30	No symptoms	None
18	30	No symptoms	Mild exercise
19	10	CONVULSED	Mild exercise
20	30	No symptoms	Mild exercise
21	20	Lip twitching	Mild exercise
22	30	No symptoms	Mild exercise
23	30	No symptoms	Mild exercise
24	30	No symptoms	Mild exercise
25	27	CONVULSED	Mild exercise
26	30	No symptoms	Work with weights
27	30	No symptoms	Mild exercise
28	30	No symptoms	Mild exercise
29	20	Lip twitching	None
30	23	CONVULSED	Mild exercise
31	27	CONVULSED	Mild exercise
32	30	Lip twitching	Mild exercise
33	30	No symptoms	Mild exercise
34	18	CONVULSED	None
35	19	CONVULSED	None
36	30	No symptoms	Mild exercise
37	30	CONVULSED	Mild exercise
38	25	Lip twitching	None
39	30	No symptoms	Mild exercise
40	29	Lip twitching	None
41	18	Lip twitching	None
42	30	No symptoms	None
43	30	No symptoms	None
44	30	No symptoms	Mild exercise
45	25	Lip twitching	Mild exercise
46	30	No symptoms	Mild exercise
47	30	No symptoms	None
48	30	No symptoms	Mild exercise
49	24	CONVULSED	None
50	30	No symptoms	None
51	20	CONVULSED	None
52	30	Lip twitching	None
53	30	No symptoms	None

No.	Time	Symptoms	Work done
54	30	No symptoms	None
55	30	No symptoms	None
56	29	CONVULSED	None
57	30	No symptoms	None
58	30	No symptoms	None
59	27	CONVULSED	Mild nausea
60	30	No symptoms	None
61	30	No symptoms	None
62	30	No symptoms	None
63	30	No symptoms	None
64	30	No symptoms	None
65	30	No symptoms	None
66	30	No symptoms	None
67	30	No symptoms	None
68	30	No symptoms	None
69	22	CONVULSED	None
70	29	CONVULSED	None
71	16	CONVULSED	None
72	30	Lip twitching	None
73	30	No symptoms	None
74	29	CONVULSED	None
75	30	No symptoms	None
76	30	No symptoms	None
77	30	No symptoms	None
78	30	No symptoms	None
79	20	CONVULSED	None
80	30	No symptoms	None
81	30	No symptoms	None

82	7	CONVULSED	None
83	30	No symptoms	None
84	20	Severe lip twitching	None
85	28	CONVULSED	None
86	20	CONVULSED	None
87	22	Lip twitching	None
88	10	Lip twitching	None
89	18	Lip twitching	None
90	30	No symptoms	None
91	31	Nausea	None
92	30	No symptoms	None
93	24	CONVULSED	None
94	30	No symptoms	None
95	30	No symptoms	None
96	15	CONVULSED	None
97	12	CONVULSED	None
98	17	Lip twitching	None
99	18	Lip twitching	None
100	11	Lip twitching	None

TABLE 4

TIMES OF TOLERANCE OF FIVE SUBJECTS AT VARIOUS DEPTHS IN THE WET

	50FT.		60FT.		70FT.		80FT.		90FT.		100FT.	
	DEPTH OF FEET OF SEAWATER											
	Time in Mins	Day and Symptoms	Time in Mins	Day and Symptoms	Time in Mins	Day and Symptoms	Time in Mins	Day and Symptoms	Time in Mins	Day and Symptoms	Time in Mins.	Day and Symptoms
WHITTINGTON	58	23rd Severe lip twitching	61	5th Bad lip twitching	21	30th Lip twitching	8	37th Lip twitching	11	41st Lip twitching	12	28th Lip twitching
BROWN	44	26th Severe lip twitching	19	5th Convulsed	34	33rd Lip twitching	37	37th Lip twitching	12	40th Lip twitching	15	28th Lip twitching
HARMAN	112	22nd Severe lip twitching	76	6th Severe lip twitching	44	29th Lip twitching	26	35th Lip twitching	25	44th Lip twitching	19	27th Lip twitching
FRASER	120	19th No symptoms	37	7th Lip twitching	74	34th Lip twitching	45	36th Lip twitching	18	41st Lip twitching	19	29th Lip twitching
DICKIE	121	20th No symptoms	12	9th Lip twitching	55	33rd Lip twitching	28	35th Convulsed	9	42nd Lip twitching	24	29th Lip twitching

TABLE 7

TOLERANCE OF DIVERS IN THE WET AT 30 ft RESTING

Minutes	Diver	Symptoms
3	Kirk	Lip twitching
10	Martin	Lip twitching
11	Mullaney	Lip twitching
12	Gibson	CONVULSED
15	Manton	CONVULSED
15	Whitbeam	CONVULSED
16	Mantell	CONVULSED
17	Knight	CONVULSED
17	Hinton	Lip twitching
18	Green	Slight lip twitching
19	Hill	CONVULSED
19	Hill	Lip twitching, Nausea
19	Mahons	Lip twitching
20	Donnelly	CONVULSED
20	Carpenter	CONVULSED
20	McCann	Lip twitching
22	Dodd	Lip twitching
23	Lachlan	CONVULSED
24	McLaughlin	CONVULSED
25	Sims	Lip twitching, headache
27	Liddle	CONVULSED
28	Gray	CONVULSED
28	Powell	Slight lip twitching, vertigo

Minutes	Diver	Symptoms
28	Smith	CONVULSED
29	Symington	CONVULSED
29	Woolcott	CONVULSED
35	Ward	Lip twitching, nausea
35	McAtamney	Lip twitching and loss of consciousness
35	Robertson	Lip twitching
44	Brown	Slight lip twitching
59	Whittington	Lip twitching and vertigo
69	Shields	CONVULSED
85	Smith I	Sudden lip twitching
90	Rogers	Lip twitching and nausea
90	Derrick	Coughing
99	Wallis	Lip twitching
112	Harman	Severe lip twitching
120	Fraser	No symptoms
120	Warren	No symptoms
121	Dickie	No symptoms

Total No. of Divers - 40.

TABLE 8

TOLERANCE OF DIVERS IN THE WET AT 60 ft RESTING

Minutes	Diver	Symptoms
2	Kirk	Lip twitching and vertigo
8	Ward	Lip twitching
9	Hutton	Lip twitching
12	Dickie	Lip twitching
13	Sims	Lip twitching
15	Mulberry	Lip twitching
16	McAtamney	Vertigo
19	Brown	CONVULSED
19	Robertson	Lip twitching
23	Martin	Lip twitching
24	Wallis	Lip twitching
25	Warren	CONVULSED
37	Rogers	Severe lip twitching
37	Fraser	Lip twitching
40	Derrick	Lip twitching
73	Whittington	Severe lip twitching
76	Harman	Lip twitching and body tremors
80	Smith II	Severe lip twitching

Total No. of Divers – 18

TABLE 9

TOLERANCE OF DIVERS IN THE WET AT 90 ft RESTING

Minutes	Diver	Symptoms
15	Kirk	Lip twitching
4	Mulberry	Lip twitching
8	Martin	Vertigo and Nausea
10	Wallis	Lip twitching and vertigo
11	Robertson	Lip twitching
12	Sims	Lip twitching
14	Brown	Slight lip twitching and dyspnoea
15	Liddle	CONVULSED
15	Powell	Lip twitching
17	Clarkson	Lip twitching
17	Symington	CONVULSED
19	Whittington	Lip twitching
20	Baldwin	Lip twitching
23	Hill	Syncope
26	Rogers	Lip twitching
26	Herrett	Lip twitching
32	Miller	CONVULSED
38	Ward	Lip twitching
39	McAtamney	Vertigo, feeling of exhaustion, dazed.
43	Warren	Slight lip twitching
44	Harman	Lip twitching
55	Dickie	Lip twitching
74	Fraser	Slight lip twitching
77	Derrick	Slight lip twitching

Total No. of Divers - 24

TABLE 10

TOLERANCE OF DIVERS IN THE WET AT 80 ft RESTING

Minutes	Divers	Symptoms
2	Martin	Vertigo
3	Kirk	Lip twitching
4	Mulberry	Lip twitching, vertigo
7	Hutton	Lip twitching
8	Smith I	Lip twitching
8	Whittington	Lip twitching
8	Wallis	Lip twitching
9	Robertson	Severe lip twitching
11	Woolcott	Severe lip twitching, nearly out
13	Clarkson	Lip twitching
13	Sims	Lip twitching, headache
13	Smith II	Spasmodic respiration, manual tremors
13	Ward	Lip twitching, headache
14	Baldwin	Lip twitching
15	Herrett	CONVULSED
17	Hill	CONVULSED
18	McAtamney	Vertigo, weariness
20	Derrick	Lip twitching
22	Warren	Lip twitching
25	Harman	Lip twitching
27	Rogers	Lip twitching, nausea
27	Powell	CONVULSED

Minutes	Divers	Symptoms
28	Dickie	Coughing, CONVULSED
33	Ware	CONVULSED
35	Brown	Lip twitching
44	Fraser	Slight lip twitching, dyspnoea
75	Miller	Lip twitching

Total No. of Diver – 27.

TABLE 11

TOLERANCE OF DIVERS IN THE WET AT 90 ft RESTING

Minutes	Diver	Symptoms
2	Kirk	Lip twitching
4	Wallis	Lip twitching
4	Mulberry	Lip twitching, body and leg tremors
7	Ward	Lip twitching
9	McAtasney	Drowsiness, CONVULSED
9	Sims	Severe lip twitching
9	Dickie	Lip twitching
11	Whittington	Lip twitching
11	Rogers	Lip twitching and vertigo
11	Robertson	Lip twitching, CONVULSED
12	Brown	Lip twitching
18	Fraser	Lip twitching
25	Harman	Lip twitching
38	Derrick	Coughing
71	Smith II	Lip twitching

Total No. of Drivers – 15

TABLE 12

TOLERANCE OF DIVERS IN THE WET AT 100 ft RESTING

Minutes	Diver	Symptoms
2	Kirk	Lip twitching
3	Wallis	Lip twitching, headache
3	Mulberry	Lip twitching, Pulsation in arms
3	Ward	Vertigo, drowsiness
4	Robertson	Lip twitching
4	Hutton	Lip twitching, vertigo
5	McAtamney	Vertigo, weakness
7	Sims	Lip twitching
7	Rogers	Lip twitching, nausea, slight vertigo
11	Brown	Severe lip twitching
12	Whittington	Lip twitching
19	Fraser	Lip twitching
24	Dickie	Lip twitching
36	Derrick	Lip twitching

Total No. of Divers – 14.

TABLE 13

ASSESSMENT OF INDIVIDUAL AND GROUP VARIATION
LOGARITHM OF DIVER'S TIME MINUS LOGARITHM OF MEAN VALUE FOR EACH DEPTH

Pressure	50 ft. under-water	60 ft. under-water	70 ft. under-water	80 ft. under-water	90 ft under-water	100 ft. under-water	90 ft. in air	Variance	Standard Deviation
Diver									
Kirk	-1.2559	-1.0038	-1.1152	-0.6603	-0.7154	-0.4717	-0.6639	0.08159	0.2856
Mulberry	-0.6910	-0.1287	-0.6892	-0.4842	-0.4143	-0.3925	-0.4879	0.03717	0.1928
Wallis	+0.2622	+0.0844	-0.2701	-0.2080	-0.4143	-0.3925	-0.0804	0.06280	0.2506
Robertson	-0.1822	0.0148	-0.2499	-0.1597	+0.0445	-0.2675	-0.0329	0.01440	0.1200
Ward	-0.1883	-0.4017	-0.2885	-0.0235	-0.1413	-0.3577	+0.0764	0.05909	0.2431
Sims	-0.3029	-0.1909	-0.2121	-0.0235	-0.387	+0.0055	+0.3840	0.05090	0.2256
McAtamney	-0.1822	-.01007	+0.2998	+0.1179	-0.0622	-0.1706	-0.0356	0.02980	0.1726
Brown	-0.0840	-0.0260	-0.1452	+0.4128	+0.0628	+0.1911	+0.2655	0.04078	0.2019
Rogers	+0.2218	+0.2634	+0.1237	+0.3019	+0.0443	+0.0055	-0.1968	0.02939	0.1714
Whittington	+0.0421	+0.5615	-0.0013	-0.2343	+0.0250	+0.2185	+0.2943	0.06495	0.2549
Dickie	+0.5281	-0.2079	+0.4491	+0.3098	+0.0887	+0.5240	+0.0631	0.05397	0.2896
Smith II	+0.1970	+0.5983	+0.3458	-0.0235	+0.8349	+0.0055	-0.2117	0.13719	0.3704
Fraser	+0.5281	+0.2692	+0.5779	+0.5110	+0.2508	+0.4092	+0.0957	0.03247	0.1802
Derrick	+0.2218	+0.2973	+0.9980	+0.1636	+0.5634	+0.6927	+0.5402	0.04308	0.2076
Mean								0.05483	0.2342

TABLE 17

DIVES IN THE WET ON OXYGEN AT 40 FT WITHOUT WORK

Name of Diver	Time in Min.	Symptoms
Donnelly	150	Nil
Brown	67	Lip twitching
Mulberry	92	Lip twitching, nausea
McAtamney	90	Lip twitching
Robertson	120	None
McAtamney	120	None
Rogers	120	None
Wallis	120	None
Allender	28	CONVULSED
Brewster	28	CONVULSED
Gibson	120	None
Kirk	18	Lip twitching
Brown	30	CONVULSED
Smith	12	CONVULSED
Ward	120	None
Kirk	18	Lip twitching
Martin	19	Lip twitching and nausea
Hunt	120	None
Hutton	29	Lip twitching
Gibson	50	Lip twitching, nausea
Hutton	120	None
Rutter	29	Lip twitching

Snelling	120	None
Kirk	27	Lip twitching
Gibson	55	Nausea and lip twitching
Seaner	120	None
Rolfe	120	None
Elsby	120	None
Symmington	150	None

TABLE 18

DIVES IN THE WET ON OXYGEN AT 35 FT WITHOUT WORK

Name of Diver	Time in Min.	Symptoms
Brown	180	None
Gibson	180	None
Kirk	30	CONVULSED
Williams	14	Lip twitching
Kirk	32	Lip twitching
Hunt	120	None
Hutton	120	None
Senner	40	Throat twitching
Elsby	120	None
Snelling	120	None
Rutter	31	Lip twitching
Sims	90	None
Smith	30	Lip twitching
Rogers	90	None
McAtamney	90	None
Ward	90	None
Scott	90	None
Martin	90	None
Wallis	90	None
Mulberry	90	None
Hutton	90	None

TABLE 19

DIVES IN THE WET ON OXYGEN TO 30 FT WITHOUT WORK

Name of Diver	Time in Min.	Symptoms
Robertson	120	None
Kirk	48	CONVULSED
Gibson	120	None
Ward	120	None
Hunt	120	None
Wallis	120	None
Rogers	120	None
Hutton	120	None
McAtamney	43	CONVULSED
Snelling	120	None
Senner	120	None
Rutter	24	Vertigo
Rolfe	120	None
Robertson	90	None
Mulberry	90	None
Sims	90	None
Kirk	90	None
Scott	90	None
Martin	90	None
McAtamney	90	None

TABLE 20

DIVES IN THE WET ON OXYGEN TO 25 FT WITHOUT WORK

NAME OF DIVER		
Robertson	Downer	Kenny
Kirk	Shean	Butt
McAtamney	Mays	Leslie
Ward	Cotter	Carr
Wallis	Morley	Locke
Gibson	Stubbs	Snelling
DeAsha	Payne	Rolfe
Merriman	A. Smith	Semer
McIlduff	Maidment	Rutter
	Elsby	

All these divers completed 120 minutes, at 25 feet, with no symptoms, except Butt, who suffered from nausea and vomiting after 44 minutes. This subject had similar symptoms when breathing oxygen at atmospheric pressure.

TABLE 21

OXYGEN POISONING AT 50 FT. IN THE WET WITH WORK

Subject	Times in min.	Symptoms	Total Work done in ft. lbs	Control without Work	
				Time in Min.	Symptoms
Elsby	8	Lip twitching	12,801	120	None
Elsby	27	Lip twitching	28,105	120	None
Elsby	33	Lip twitching	31,500	–	–
Friels	22	Lip twitching	25,200	21	Nausea Vertigo
Friels	–	–	–	28	Nausea
Grabham	15	Lip twitching Vertigo	14,630	10	Lip twitching
Grabham	–	–	–	12	Lip twitching
Gibson	8	Lip twitching	5,101	19	Body tremors
Gibson	9	Lip twitching	11,068	20	Lip twitching
Gibson	10	Lip twitching	5,740	12	Lip twitching
Hunt	53	Lip twitching	53,033	120	None
Hunt	60	Vertigo	56,017	67	Lip twitching
Hunt	63	Lip twitching	70,350	–	–
Hutton	13	Lip twitching	22,137	20	Body tremors
Hutton	22	Lip twitching	29,741	20	Lip twitching
James	64	Lip twitching	66,640	120	None
Kirk	1	Lip twitching	1,480	73	Lip twitching
Kirk	3	Vertigo	3,712	4	Lip twitching
Kirk	4	Lip twitching	4,372	7	Lip twitching
McAtamney	10	CONVULSED	28,328	42	Lip twitching
McAtamney	10	Vertigo	10,395	20	Vertigo

Subject	Times in min.	Symptoms	Total Work done in ft. lbs	Control without Work	
				Time in Min.	Symptoms
McAtamney	9	Vertigo	5,390	38	Vertigo
Mulberry	6	Lip twitching, nausea	15,134	12	Lip twitching
Robertson	8	Lip twitching	18,826	120	None
Robertson	7	Lip twitching, nausea	10,202	120	None
Robertson	20	Lip twitching	35,011	–	–
Rogers	16	Vertigo	28,216	120	None
Rogers	32	CONVULSED	61,710	101	Lip twitching
Rogers	48	Pain in chest (probably not O_2	50,380	121	None
Rogers	24	Lip twitching, Vertigo	25,130	–	–
Rolfe	43	Lip twitching	45,141	120	None
Rolfe	38	Lip twitching	41,404	120	None
Rolfe	18	Lip twitching	17,010	–	–
Rutter	5	Lip twitching	6,545	10	Lip twitching
Rutter	12	Lip twitching	19,346	–	–
Senner	3	Body tremors	7,988	120	None
Senner	–	–	–	43	CONVULSED
Senner	6	CONVULSED	6,256	8	Lip twitching
Senner	–	–	–	5	Throat tremors
Senner	4	Throat tremors, nausea	3,920	–	–
Sims	22	Lip twitching	26,516	26	Lip twitching
Snelling	37	Lip twitching	49,761	120	None
Snelling	56	CONVULSED	48,895	–	–
Wallis	11	Lip twitching	28,918	99	Lip twitching, nausea
Wallis	12	Lip twitching	22,618	28	Lip twitching, vertigo
Wallis	16	Lip twitching	34,168	65	Lip twitching
Wallis	40	Lip twitching vertigo, nausea	44,590	–	–
Ward	13	Nausea	12,993	35	Nausea
Ward	29	Nausea	42,446	79	Lip twitching
Ward	33	Nausea	–	39	Lip twitching

TABLE 22

OXYGEN POISONING AT 40 ft. IN THE WET WITH WORK

Subject	Time in Min.	Symptoms	Total Work done in ft. lbs.	Control without Work	
				Time in Min.	Symptoms
Elsby	15	Lip twitching	22,137	120	None
Elsby	60	None	81,427	–	–
Elsby	120	None	113,400	–	–
Friele	45	Vertigo, nausea	38,500	–	–
Gibson	10	Lip twitching	9,336	120	None
Gibson	34	Lip twitching, body tremor	50,435	30	Lips, nausea
Gibson	13	Lip twitching	12,110	–	–
Grabham	16	Lip twitching	16,100	–	–
Hunt	60	None	93,940	120	None
Hunt	30	Nausea	44,563	–	–
Hunt	48	Lip twitching	99,099	–	–
Hunt	120	None	187,790	–	–
Hutton	22	Lip twitching	38,981	120	None
Hutton	38	Lip twitching	43,601	–	–
James	120	None	97,020	–	–
Kirk	8	Vertigo	7,837	24	Lip twitching
Kirk	6	Lip twitching	6,765	18	Lip twitching
Kirk	6	Lip twitching	4,200	27	Lip twitching
McAlarney	7	Nausea	11,686	120	None
McAlarney	8	Vertigo	7,604	–	–
Robertson	60	None	89,801	90	None
Robertson	60	None	80,753	120	None
Rogers	60	None	109,670	120	None
Rogers	120	None	141,180	90	None
Rogers	43	Nausea	78,650	–	–
Rogers	39	CONVULSED	40,040	–	–
Rogers	35	Dyspnoea	66,230	–	–
Rolfe	38	Lip twitching	40,714	120	None
Rolfe	23	Dyspnoea	17,768	–	–
Rolfe	41	Vertigo	27,790	–	–
Rutter	6	Vertigo	5,293	29	Lip twitching
Rutter	3	Dyspnoea	1,905	–	–
Senner	5	Throat tremors	8,566	120	None
Senner	17	Vertigo	22,426	5	Lip twitching
Senner	9	Lip twitching, throat tremors	10,220	–	–
Snelling	60	None	79,046	120	None
Snelling	60	None	63,525	–	–
Wallis	60	Lip twitching	92,592	120	None
Wallis	30	Lip twitching	54,092	90	None
Wallis	35	Nausea	38,981	–	–
Wallis	57	Lip twitching	131,670	–	–
Wallis	64	Lip twitching	104,896	–	–
Wallis	28	Nausea	29,750	–	–
Ward	30	Vomiting	31,281	120	None
Ward	35	Lip twitching	38,981	90	None
Ward	46	Nausea	63,070	–	–

TABLE 23

OXYGEN POISONING AT 30 ft. IN THE WET WITH WORK

Subject	Time in Minutes	Symptoms	Total Work done in ft. lbs.	Control without Work	
				Time in Minutes	Symptoms
Friels	51	Nausea	40,110	–	–
Gibson	39	Vertigo	24,736	120	None
Gibson	61	Nausea	74,270	–	–
Kirk	34	Lip twitching Vertigo	–	48	CONVULSED
Kirk	37	Vertigo	24,640	90	None
McAtominey	19	Vertigo	22,715	43	CONVULSED
Rogers	60	Vertigo, nausea	114,950	120	None
Rogers	24	Vertigo	56,107	–	–
Rogers	120	None	132,180	–	–
Rutter	17	Lip twitching	24,447	24	Vertigo
Sennet	10	CONVULSED	14,533	120	None
Sennet	21	Lip twitching	19,880	–	–
Wallis	120	None	126,770	120	None
Ward	120	None	129,150	120	None

TABLE 24

OXYGEN POISONING AT 28 ft. IN THE WET WITH WORK

Subject	Time in Min.	Symptoms	Total Work done in ft. lbs.	Control without Work	
				Time in Min.	Symptoms
Friels	120	None	169,190	–	–
Gibson	60	Vertigo	61,118	120	None
Gibson	120	None	126,400	–	–
Kirk	42	Lip twitching	37,785	120	None
Kirk	120	None	119,420	–	–
Sennet	28	Lip twitching	36,211	120	None
Sennet	54	Lip twitching Vertigo	56,017	–	–
Sennet	120	None	89,110	–	–
Ward	60	None	60,076	120	None
Wallis	60	None	111,833	120	None
Rogers	60	None	148,449	60	None
Robertson	60	None	130,322	120	None
McAtasney	60	None	111,553	120	None
Hutton	54	Vertigo	94,806	–	–
Rolfe	60	None	81,138	120	None
Seelling	60	None	160,446	120	None
Bixby	60	None	227,150	120	None
Rutter	60	None	91,630	120	None

APPENDIX 3

SYMBOLS

These are based on those recommended by Pappenheimer (Fed. Proc.,1950, 9, 602).

Primary Symbols: are printed in italics and are as follows:

V	volume of gas or blood	F	Fractional concentration of gas
V	gas volume per unit time	f	Frequency of respiration per minute
P	pressure	D	equivalent depth

The symbol P is used in several ways:

(a) Describing atmospheric or ambient pressure
(b) When representing partial pressure exerted by a designated gas in a mixture of gases.
(c) When representing tension of a designated gas dissolved in a liquid.

Suffixes: qualify the primary symbol and define the anatomical site and/or substance to which the measurement refers.

I	Inspired gas	B	Barometric pressure
E	Expired gas	AMB	Diver's ambient pressure
A	Alveolar gas	a	Arterial blood
T	Tidal gas	v	Venous blood
ET	End-tidal gas	$\bar{v}$	Mixed venous blood

Suffixes are printed in Roman type, capitals when in gas phase and lower case when in liquid phase.

DEPTH AND PRESSURE

Atmospheres (or bars) are employed to define pressure, ATA for atmospsheres absolute, ATS for atmospheres above one atmosphere.

Metres or feet of sea water (msw, fsw) are used when giving the depth of the diver.

These conventions are not strictly observed in pressure work.

Useful 'running' approximations are:

10 msw = 33 fsw = 1 ATS = 2 ATA = 2 bar
30 msw = 100 fsw = 3 ATS = 4 ATA = 4 bar

Finally, units of volume or pressure are specified throughout and are always those used by the author whose work is being described or reviewed.