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.

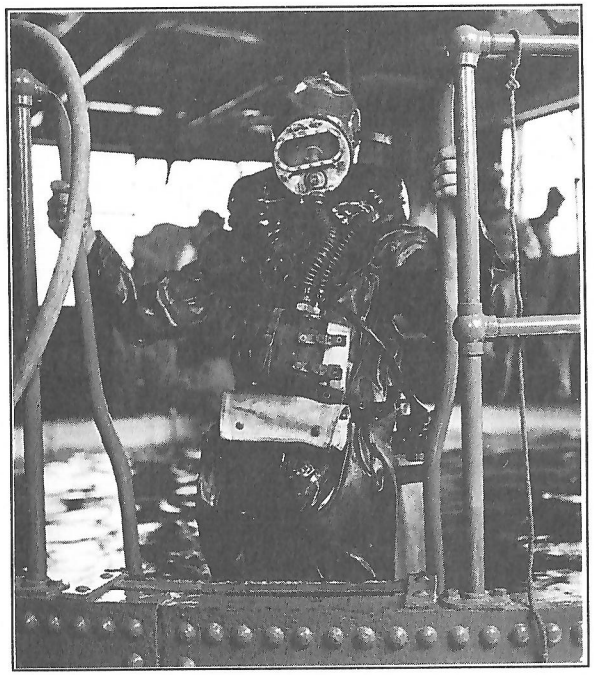
End (1937) appears to be one of the first, if not the first, to report that there was a "marked absence of psychological change" breathing oxygen-helium mixture at considerable pressures. His colleague, Nohl, used such a mixture in a world record dive to 420 fw in Lake Michigan in 1937 (End, 1938). This 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

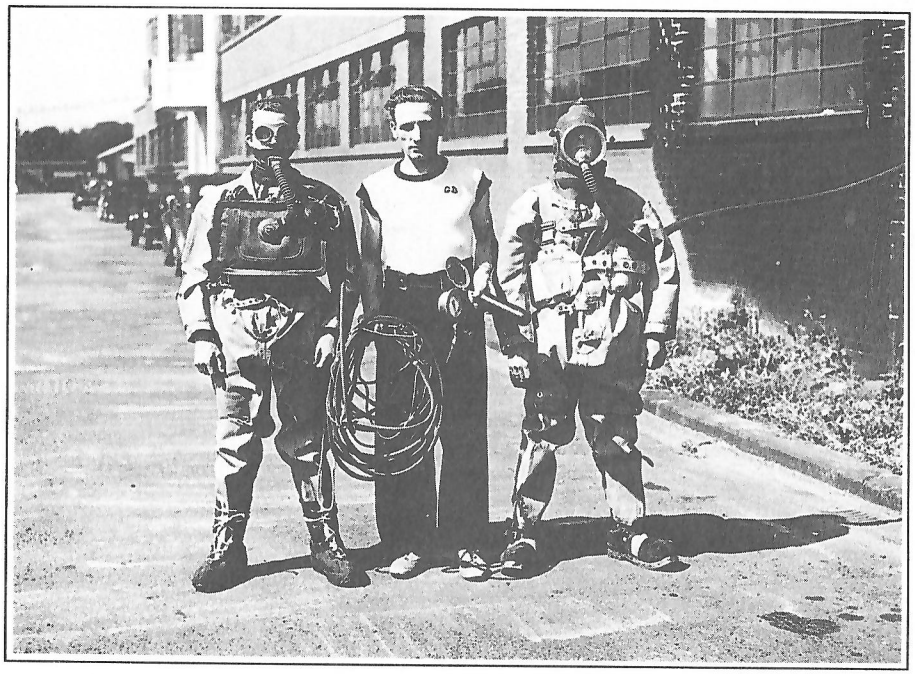
oxygen uptake, first in dry chambers and then underwater. Gases were sampled from the counterlung during various stages of these dives to check against calculated figures and to be sure of the partial pressure of oxygen and nitrogen being breathed in each experimental dive. Both non-toxic and deliberately 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 1/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.

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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_3 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.

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