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 O.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 O.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)

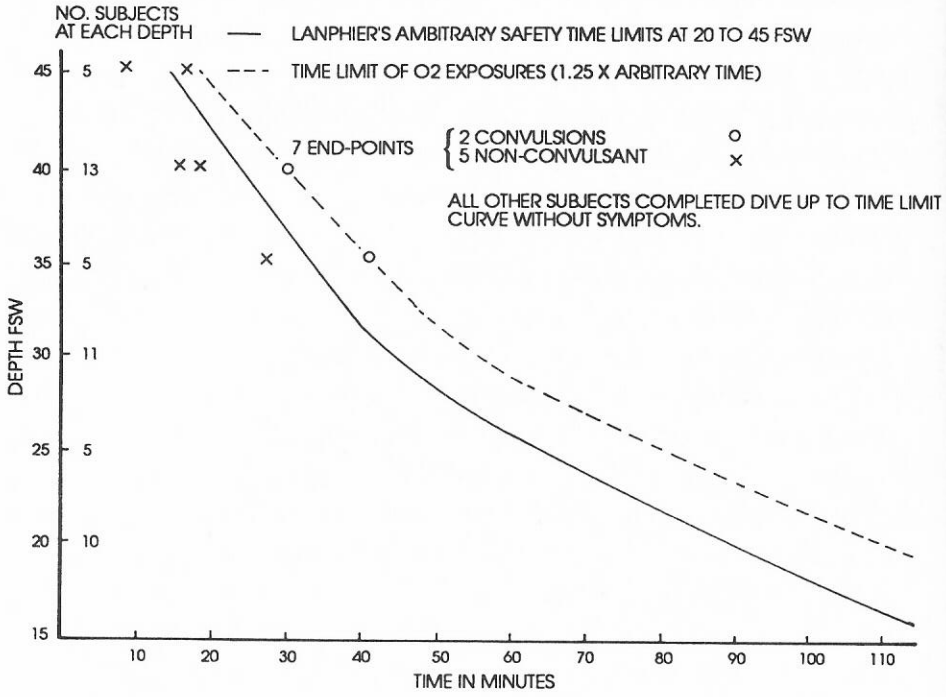

FIG. 19: Illustrating Lanphier's series of dives on pure oxygen between 20 and 45 fsw. The ambitiary time limits, the maximal exposure allowed and number of subjects at each depth are shown. Times of occurrence of symptoms causing cessation of exposure (end-point) are also plotted.

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 $\pm$ 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).

US NAVY OXYGEN DEPTH-TIME WORKING LIMITS 1947 TO 1986

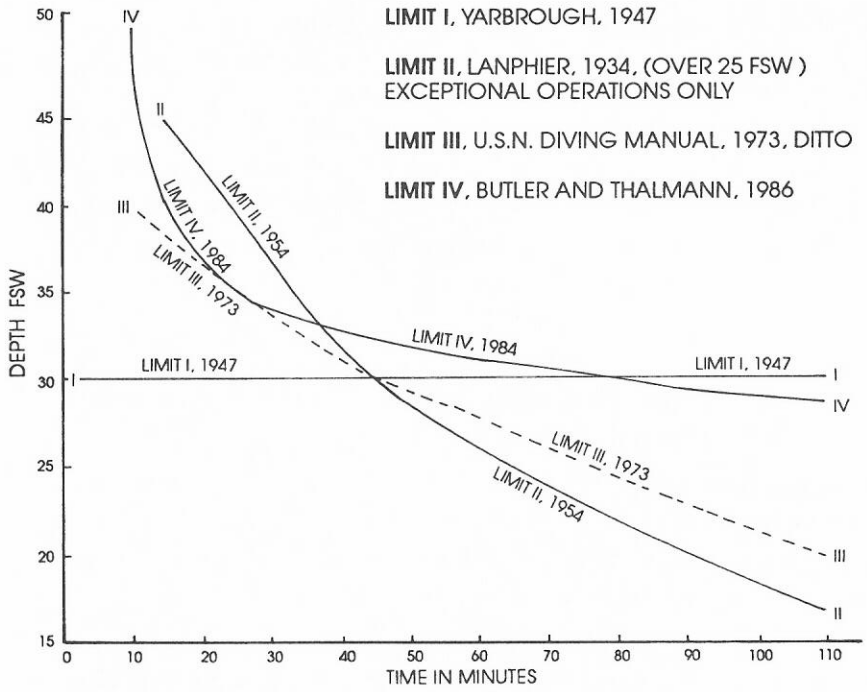

FIG. 20: Illustrating U.S. Navy oxygen depth-time working limits (with exercise). Note that limits II and III could only be used over 25 fsw in exceptional operational conditions. This does not apply to limit IV (Butler & Thalman, 1986).

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.

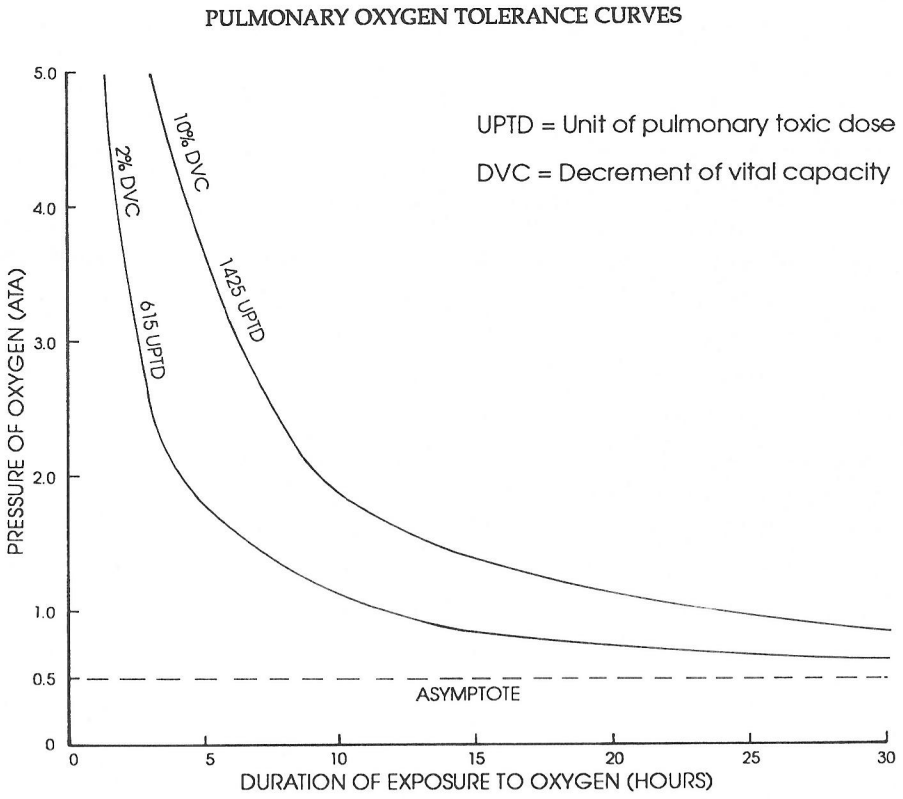

FIG. 21: Hyperbolia curves showing time of exposure to oxygen to cause 2 and 10% decrement of vital capacity (50% of group) at different pressure. Normal male subjects (after Clark & Lambertson, 1971).

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

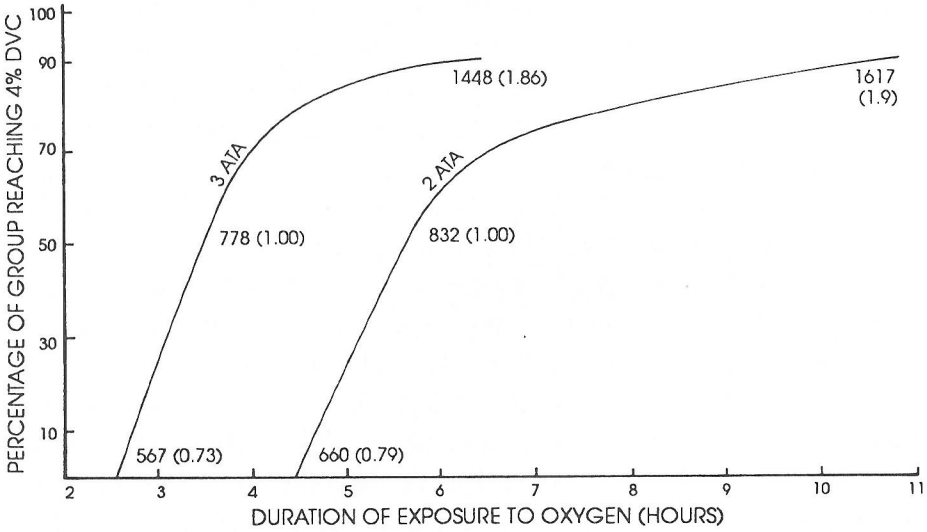

FIG. 22: Curves showing distribution of pulmonary oxygen tolerance in a group of normal men at 2 and 3 ATA as measured by time to achieve 4% decrement of vital capacity (DVC). Dosage UPTD to achieve 4% DVC shown for 10%, 50% & 90% of the group at each pressure. Ranges in parenthesis. Average dose as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